

DECEPTION AND EXERCISE PERFORMANCE DURING REPEATED
4-KM CYCLING TIME TRIALS

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Submitted to the faculty of the University Graduate School
in partial fulfillment of the requirements for the degree
Master of Science
in the Department of Kinesiology
Indiana University
July 2013

Accepted by the Graduate Faculty, Indiana University, in partial fulfillment of the requirements for the degree of Master of Science in Kinesiology.

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26 July 2013

ACKNOWLEDGMENTS

The path to completing this thesis was a little longer than I expected and took a few extra twists and turns as well. Despite this, its completion brings me great pride and this would not have been possible without the help and contributions of many friends and colleagues. I would like to acknowledge and thank a few of them here.

First and foremost, I must thank the members of my thesis committee, Drs. Tim Mickleborough, Rob Chapman, and Jack Raglin, without whom this thesis would not have been possible. Dr. Mickleborough has served as my academic advisor, committee chair, and has been a friend for years before I began my studies here at IU. I am indebted to him for his guidance, help, and mentorship and I hope the completion of this thesis brings him some degree of gratification. Drs. Mickleborough and Raglin were instrumental in the design of the study and have given me steadfast guidance and direction in both this study and my graduate studies in general. Dr. Chapman has given me consistent, pragmatic advice and guidance and his work in maintaining and updating the research laboratory made it possible for me to complete my project in a timely manner. The completion of this study and preparation of this manuscript is a testament to the commitment and guidance of these committee members, so I hope they can view this with a measure of pride.

I thank Anne Stephenson, Debbie Szemcsak, Susan Provenzale-Todd, Bob O'Loughlin and all the wonderful people in the Dean's Office who helped me along the way with everything from tracking down the right forms to scheduling meeting dates, and countless other small, but important things. My academic career would have been mired by paperwork and many hours would have been spent searching for answers without their help, so I am indebted to them for their assistance.

Bloomington, IN has been my home for over 25 years and I have always had some affiliation with Indiana University. Since beginning graduate school here however, my passion for IU has never been stronger. The faculty, staff, and fellow students in the Department of Kinesiology and School of Public Health have made me feel welcome and at home since I began my studies here, so I am grateful to have been able to meet such wonderful people. I would be remiss in my acknowledgments if I did not thank my fellow graduate students, with whom I have learned, taught, discussed, conducted research, laughed, and commiserated with, among other things. It has been an honor to share their company and I continue to enjoy watching my predecessors grow and succeed as physiologists in the hopes that I can follow in their footsteps.

My family has been a source of constant support and patience and I am truly blessed to have them by my side throughout this life. My parents, Bruce and Hwa-Mei Shei have been unwavering in their support of me, despite my obstinate and sometimes foolhardy personality. I am continually impressed with my mother's ability to strike up a conversation with anyone and am still puzzled as to how everyone I meet seems to know her. I hope I continue to make her proud when she speaks with people that I've encountered. My father's intellect and careful attention to detail is unsurpassed and I am constantly in awe of both to this day. His example has given me a goal to aspire towards, which I hope I someday will achieve. Together, they have motivated my brother and I to strive to succeed in all that we do, and for that we are both eternally grateful.

Finally, I thank my best friend and companion Amy, who has been by my side throughout my graduate studies. Her love and support has enabled me to succeed in all that I do. I am truly blessed to have her as a lifelong companion and am thankful for each day that we have together.

ABSTRACT

Ren-Jay Shei

DECEPTION AND EXERCISE PERFORMANCE DURING REPEATED 4-KM CYCLING TIME TRIALS

INTRODUCTION: Cyclists have been shown to improve their performance in a simulated 4-km laboratory based time trial when given feedback derived from a prior performance which is surreptitiously augmented. Presently, it is unknown whether or not these performance gains are persistent after the subjects are informed of the deception. **PURPOSE:** The aim of this study was to investigate whether or not performance gains achieved through deception persist after the deception was revealed. A secondary aim of this study was to assess whether the subjects' pacing strategy changed after the deception was revealed. **METHODS:** The subjects were trained competitive cyclists. All subjects who were admitted into the study completed a total of four (4) simulated 4-kilometer cycling time trials comprising of a familiarization trial, baseline trial (BAS), deception trial (DEC), and knowledge of deception trial (KDE) performed on separate occasions. In the DEC and KDE trials, subjects competed against an on-screen avatar set to 102% of their baseline average power output. Time to completion, average power output, mean power output for each 0.5-km segment of the distance covered, and change in blood lactate concentration from pre- to post- time trial for each time trial were recorded. **RESULTS:** Subjects who completed the DEC trial faster compared to BAS trial also completed the KDE trial faster compared to the BAS trial ($F = 13.61, p = 0.003$), but time to completion in DEC and KDE trials were not different in these subjects (95% CI = -3.3 to 4.3s). Subjects who did not complete the DEC trial faster than the BAS trial also demonstrated differences in time to completion ($F = 17.31, p = 0.003$), with the KDE trial being completed faster than the DEC trial

(95% CI = 5.3 to 17.5 s), but not the BAS trial (95% CI = -6.3 to 15.0 s). Analysis of the pacing strategy adopted by subjects revealed no differences between trials for subjects who improved in the DEC trial ($F = 1.53, p = 0.238$), but significant differences were observed in subjects who did not improve in the DEC trial ($F = 8.91, p < 0.001$). **CONCLUSIONS:** Trained cyclists whose performance improves upon receiving surreptitiously augmented feedback during simulated 4-km time trials are able to retain their performance gains once the deception is revealed. They do not appear to adopt a different pacing strategy in either of the improved trials (DEC and KDE).

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CHAPTER ONE

INTRODUCTION

Fatigue is a condition in which the ability to maintain force or power output during sustained or repeated muscle contraction is compromised, or the capacity of a muscle to do work is lost (Asmussen 1979, Bigland-Ritchie, Johansson et al. 1983). Although it is well characterized that fatigue can be induced by strenuous exercise (Chaffin 1973, Asmussen 1979, Gandevia 1998), the exact mechanisms through which fatigue is developed are poorly understood (Enoka and Stuart 1992, Fuglevand 1996, Gandevia 1998). Several contributing factors have been suggested, including biochemical changes in the metabolic milieu (Amann 2011), such as acidosis (Allen, Lamb et al. 2008) and increased inorganic phosphate levels (Westerblad, Allen et al. 2002), and decrease in motoneuron firing rate (Fuglevand 1996). However, neither the relative contributions nor the interactions of these factors have been elucidated. Nevertheless, it is clear that the development of fatigue is due to a failure to maintain homeostasis, whether manifested in the metabolic milieu of the working muscle(s) or in the central nervous system.

Recently a novel model of integrative central control has been proposed as a regulatory mechanism for the maintenance of homeostasis during exercise. This model proposes that a ‘central governor,’ existing in the subconscious brain, integrates conscious input and afferent information from the periphery and consequently modulates neural output in order to regulate the exercise intensity and stay within homeostatic limits (Noakes, St Clair Gibson et al. 2004, St Clair Gibson and Noakes 2004). Peripheral information such as pH changes, phosphagen depletion, and temperature changes serve as afferent signals back to the central governor which are integrated into a response that results in a “resetting” of power output and/or speed, known as

“teleoanticipation” (Ulmer 1996). The result is an alteration in neural drive in order to maintain homeostasis. According to the models, during voluntary maximal exercise (e.g., a maximal cycling time trial) the central governor selects a metabolic set point at a relative maximum, maintaining a metabolic reserve, which recent studies have shown can be accessed through deception of the central governor (Stone, Thomas et al. 2012).

The superior performance with deception has been attributed to an increase in non-aerobic energy contribution during the latter stages of a time trial (TT). Similarly, improvements in time trial performance have been shown when competition is present as compared to no competition (Corbett, Barwood et al. 2012) and the improvement in performance was also attributed to a greater non-aerobic energy contribution. These data are consistent with the model of a metabolic reserve and suggest that access to this reserve is strictly regulated by the central nervous system. By using means such as deception and head-to-head competition, it is possible to “trick” the regulator (which may arguably serve as a central governor) into allowing increased non-aerobic metabolism, which enables higher power output during a self-paced maximal TT.

This increase in power output and non-aerobic energy output that leads to decreased time to completion of a cycling TT also increases the perceived exertion of subjects. A previous study has shown that the average rating of perceived exertion (RPE) for a 4-kilometer cycling TT is higher in a deception condition (102% of baseline average power output) as compared to baseline. This suggests that at some point during the time trial, the subject becomes aware of an increased exertion and knowingly continues to exert a greater effort until completion of the time trial. The perception of an increase in effort further supports the notion that previous “maximal” TT efforts occur at some relative maximum, keeping a degree of reserve which is not accessible under normal conditions.

The theory that a metabolic reserve is kept is an enticing one since it can explain in part how exercise is regulated to maintain homeostatic limits. Conversely, if no such reserve existed, then exercise could continue to the point of catastrophe. Catastrophic events such as hyperthermia and ischemia can be life-threatening conditions in addition to impairments to exercise capacity. Clearly the regulation of exercise in order to prevent such catastrophic events is an important mechanism that ensures the well-being of an athlete. By keeping a reserve at all times and limiting “maximal” exercise to a relative maximum, the safety of the athlete is maintained. Figure 1 illustrates the proposed central governor model of exercise regulation, which fits with the model of a metabolic reserve.

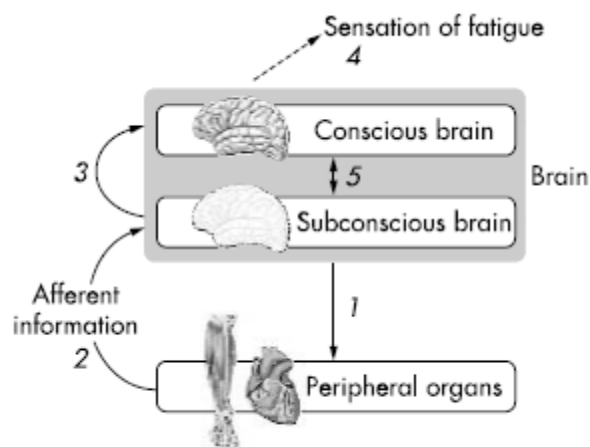


Figure 1. The central governor model of exercise regulation. (St Clair Gibson and Noakes 2004)

The existence of such a protective reserve poses some additional questions however. For example, in what conditions may this reserve be accessed? Clearly, deception can enable a person to access this reserve, but what other conditions allow access to this reserve – perhaps life-threatening situations or dire need? How is access to this reserve controlled? Furthermore, why would such a reserve exist in the body? This occurrence would seem to be superfluous,

adding one more factor to the already delicate balance of human physiology. The increased stress of keeping a metabolic reserve, e.g., the need to always maintain an available reserve pool of substrates for non-aerobic metabolism, would seem to exacerbate the challenges to maintaining bioenergetic homeostasis.

Undoubtedly it is clear that there are many questions left to be answered about the existence of a metabolic reserve and, more broadly, how homeostasis is maintained during exercise. The mechanisms leading to the development of fatigue could partially answer some of these questions and thus it becomes important to continue to try to understand how this phenomenon develops, and the mechanisms that contribute to fatigue. The development of fatigue is also closely linked to the pacing strategy that cyclists choose to adopt when performing a maximal, self-paced time trial effort.

Pacing strategy is an important component of exercise since it determines the allocation of factors such as effort and metabolic resources during exercise (Abbiss and Laursen 2008). The pattern by which bioenergetic resources are expended during an exercise bout is a contributing factor to performance in an exercise task (Hettinga, De Koning et al. 2006). Previous research has described numerous pacing strategies such as all-out, even, negative, positive, parabolic, and variable (Abbiss and Laursen 2008). Pacing strategy in laboratory-based 4-kilometer cycling time trials has been shown to be consistent between several trials, with no difference in average power output (Stone, Thomas et al. 2011). A typical profile of pacing during a 4-km cycling TT illustrates this point, and is shown in Figure 2.

The presence of other factors such as head-to-head competition (Corbett, Barwood et al. 2012) and accurate feedback (Mauger, Jones et al. 2009) have been shown to affect the pacing strategy that is adopted. As a result, the performance during a cycling time trial is improved,

suggesting the selection of a more optimal pacing strategy. Deception has been previously shown to affect pacing strategy, and the improvement in performance may have been a partial result of a more optimal pacing strategy (Stone, Thomas et al. 2012), therefore it is important to continue to investigate the effects of deception on both exercise performance and pacing strategy.

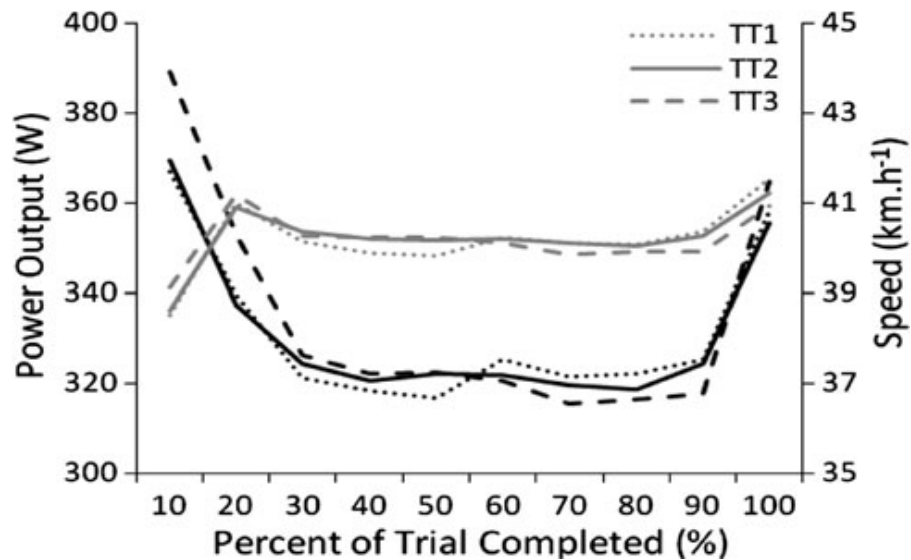


Figure 2. Power output and speed vs. percent of trial completed. Mechanical power output during the trial is indicative of the pacing strategy adopted during the trial. In this case, it is a “reverse-J” shape with an initial peak followed by a steady drop and then an end-sprint.(Stone, Thomas et al. 2011)

The improvement of exercise performance and optimization of time trial performance during a deception condition has been characterized and attributed to an increase in non-aerobic energy contribution (Stone, Thomas et al. 2012), but to date no study has evaluated performance in subsequent trials after subjects have been informed of the deception. As previously discussed,

exercise during a “maximal time trial” actually occurs at a relative maximum, with a metabolic reserve that is kept, which can be accessed using deception. In accordance with the central governor model of exercise regulation, this relative maximum is a protective mechanism which helps to regulate whole-body homeostasis by continually monitoring afferent signals, conscious input, and other factors and continually modifying central motor drive in order to maintain homeostasis. Following a deception trial in which subjects improved their performance in a 4-km cycling time trial, no homeostatic catastrophes were observed, suggesting that perhaps the presumed “central governor” may increase the relative maximum at which an individual can exercise.

Statement of the Problem

Time to completion and mean power output during a 4-kilometer cycling time trial can be improved when subjects are deceived into believing they are competing against an avatar of their own best performance, when in reality they are competing against an avatar set to 102% of their baseline mean power output. This improvement has been suggested to be the result of an increased non-aerobic energy contribution, made possible via access to a “metabolic reserve.” To date, no published research has evaluated whether or not this improvement in performance is perpetuated in subsequent trials when subjects are informed of the deception. Similarly, there is no published research documenting the pacing strategy in subsequent trials after knowledge of deception.

Purpose of the study

The purpose of this study was to evaluate whether or not the improvement in performance and changes in pacing strategy continue after subjects are informed of deception during a previous cycling time trial. The primary aim of the present study was to identify

whether time to completion remains shorter and whether average power output remains increased when subjects have knowledge of deception during a previous trial. A secondary aim was to identify whether or not changes in pacing strategy are perpetuated when subjects have knowledge of deception during a previous trial.

Delimitations

Trained male competitive cyclists with experience in cycling time trials were recruited. Data collection was conducted in a laboratory setting where such environmental conditions as temperature, humidity, and barometric pressure remain constant. Exercise tests were performed on an electromagnetically braked cycle ergometer with seat height and pedal selection set to the subjects' preference. A habituation trial was conducted to minimize variability during the experimental time trials. Subjects were asked to refrain from consumption of caffeine and alcohol for 24 hours prior to each testing session. Subjects were also asked to maintain their normal diet and training routines, and to abstain from strenuous exercise in the 24 hours prior to each testing session. Testing was conducted at the same time of day to minimize circadian variation.

Limitations

Each subject's competitive cycling experience varied, and in particular their skill in 4-kilometer time trials. When the subjects were informed that they were racing an avatar of their own best performances, the response may have been different than if they believed they were competing against another cyclist. The position on the cycle ergometer was adjusted to fit subjects as best as possible, but some subjects may not have been able to attain an optimal fit, which may have induced biomechanical impairments to their performance. The training regimen of subjects was not controlled during the course of the study.

Assumptions

The training status of the subjects did not change significantly throughout the course of the study. Subjects remained on their normal diet and training regimen for the duration of the study. Subjects refrained from strenuous exercise and ingestion of caffeine and alcohol for 24 hours prior to each experimental trial. The learning effect was minimized following a habituation trial. Subjects were highly motivated to complete each time trial the shortest amount of time possible.

Hypotheses

This study was designed to investigate the following hypotheses:

Hypothesis 1: The average power output of subjects during a 4-kilometer cycling time trial after deception is revealed will be higher.

Hypothesis 2: The time to completion of a 4-kilometer cycling time trial will be shorter after deception is revealed.

Hypothesis 3: The pacing strategy adopted by the subjects after deception is revealed will reflect an end-spurt that begins sooner, and is of a larger magnitude than in the baseline trial.

Hypothesis 4: Motivation will not differ significantly between subjects.

Definition of terms

Baseline (BAS) – the initial self-paced time trial that subjects perform to establish a reference performance.

Central fatigue (CF) – a component of fatigue that develops in the central nervous system, resulting in a decrease in motor drive to the exercising muscle(s)

Central governor model (CGM) – the central governor model of exercise regulation that proposes the existence of a “central governor” (CG) in the subconscious brain which integrates

afferent input and conscious input and continually modifies central motor drive to protect whole-body homeostasis.

Deception (DEC) – a condition in which subjects race against an avatar representing 102% of their baseline average power output, but subjects are falsely informed that they are competing against an accurate baseline avatar.

Knowledge of deception (KDE) – a condition in which subjects are aware that they have been previously deceived and again race an avatar representing 102% of their baseline average power output.

Metabolic reserve (MR) – a proposed bioenergetic reserve that is kept even during “maximal” self-paced exercise.

Oxygen consumption ($\dot{V}O_2$) – the rate of consumption of a given volume of oxygen (O_2) in one minute.

Peripheral fatigue (PF) – a component of fatigue that originates in the periphery (e.g. the muscle milieu) which may directly impair muscle contraction and/or serve as an afferent signal to the brain

Power – the rate at which mechanical work is done, given by the equation:

$$Power (Watts) = Work (Joules) / Time (Seconds)$$

Pulmonary minute expired ventilation (\dot{V}_E) – the volume of gas that is expired from the lungs in one minute.

Ratings of perceived exertion (RPE) – the level of exertion that is perceived by the subject at a given point in time, rated on a scale of 6 (easiest) to 20 (hardest). (Borg 1982)

Respiratory exchange ratio (RER) – the ratio of carbon dioxide production ($\dot{V}CO_2$) to oxygen consumption ($\dot{V}O_2$), expressed as a decimal number.

Time to completion (TTC) – the time taken to complete an exercise task of a fixed distance.

Time trial (TT) – a self-paced cycling exercise bout in which subjects attempt to complete a set course in the least amount of time.

CHAPTER TWO

REVIW OF LITERATURE

Fatigue

Fatigue is a transient, reversible condition in which the ability to maintain force or power output during sustained or repeated muscle contraction is compromised. The reduced ability of the muscles to do work impairs athletic performance, making fatigue an important component of athletic performance. Unfortunately, the mechanisms of fatigue are currently poorly understood and while there are numerous factors which are known to play a role in the development of fatigue (e.g. pH changes in the metabolic milieu, changes in motoneuron firing rate), their relative contributions have not been fully characterized. What is clear is that the development of fatigue results from a failure to maintain homeostasis, which may occur in such places as the central nervous system or the metabolic milieu of the working muscles.

Numerous models of fatigue have been proposed, however historically the two predominant models are the so-called “central” and “peripheral” models. More recently, a novel model of integrative central control, known as the “central governor model”, has been proposed as a regulatory mechanism for homeostasis during exercise. While these models share some commonalities, such as the acknowledgement of such phenomena as pH changes in the metabolic milieu, they differ significantly in how the condition of fatigue actually develops. It appears that there is no global mechanism by which muscle fatigue develops (Enoka and Duchateau 2008).

Peripheral Model

As the name suggests, the peripheral model proposes that fatigue originates in the periphery, not in the central nervous system. Specifically, the biochemical conditions in the

metabolic milieu of exercising muscles contribute significantly to the development of peripheral fatigue. Of particular interest is the energy-transfer molecule adenosine triphosphate (ATP), the primary molecule used in the body as a source of energy. The hydrolysis of the γ -phosphate yields a significant amount of energy, which can then be used to do cellular work (Berg, Tymoczko et al. 2007). The products of this hydrolysis are adenosine diphosphate (ADP) and inorganic phosphate (HPO_4^- or P_i) while a byproduct of this hydrolysis is a free proton (H^+) (Keyser 2010). The re-synthesis of ATP once it has been hydrolyzed into ADP is a key factor in this model. If ATP cannot be re-synthesized at a rate equal to the demand for ATP, then this becomes a limiting factor during exercise and fatigue will develop. There are three main bioenergetic systems for ATP synthesis: the ATP-CP (or ATP-PCr) system, the glycolytic system, and the oxidative phosphorylation system. Substrate-level phosphorylation occurs to convert ADP to ATP in the ATP-CP and glycolytic systems. Neither of these systems requires oxygen to regenerate ATP. In the oxidative system, a proton gradient is generated during electron transport in the inner mitochondrial membrane (Mitchell 1961, Mitchell 1966) and the resulting proton-motive force is used to generate ATP via the F_0F_1 ATPase complex (Nakamoto, Scanlon et al. 2008). The final electron acceptor at the end of the electron transport chain is oxygen, making this process require oxygen. During intense exercise, the high demand for ATP may exceed the ability of the oxidative system to supply ATP. Increased activity of the glycolytic system can be achieved via lactate fermentation, which converts pyruvate (a product of glycolysis) into lactate and H^+ . This process simultaneously regenerates NAD^+ from $\text{NADH} + \text{H}^+$, allowing glycolysis to continue (NAD^+ is a vital substrate in glycolysis). Non-aerobic glycolysis diverts pyruvate from the Krebs cycle and further oxidation. In this condition, the $\text{NADH} + \text{H}^+$ generated in glycolysis is not shuttled across the mitochondrial membrane and does

not enter the electron transport chain since it is oxidized back to NAD^+ by the enzyme lactate dehydrogenase. Lactic acid is an organic acid and the acidic proton can freely dissociate at normal biochemical conditions to form lactate and H^+ , causing an increase in hydrogen ion concentration (and therefore lowering the pH).

Historically, the production of lactate has been blamed for the development of fatigue (Hill and Lupton 1923, Hill, Long et al. 1924, Hill, Long et al. 1924). Frequently the development of fatigue and an increase in blood lactate concentration ($[\text{B}_{\text{La}}]$) are concomitant, so this theory that lactate causes fatigue seems reasonable. Several other factors also play a role however, since elevated lactate production frequently occurs during times of high metabolic demand. High metabolic demands are the result of a high rate of ATP hydrolysis, or energy demand, usually from repeated or sustained muscle contractions. The result of these repeated or sustained muscle contractions is that other metabolites such as free inorganic phosphate (P_i), calcium, and ammonium can accumulate (Roberts and Smith 1989, Kirkendall 1990). Lactate production causes an associated increase in $[\text{H}^+]$, which has more significant implications in bioenergetic processes.

The pH of a particular environment (e.g. cytosol of a muscle fiber) has important consequences on the chemical reactions that take place in that environment. Thus, during exercise if pH decreases, then the biochemical processes that take place within exercising muscle, such as glycolysis, will be impacted by this pH change. There is considerable evidence that lower intramuscular pH can in part inhibit glycolysis (Bergstrom, Harris et al. 1971, Karlsson 1971, Gollnick and Hermansen 1973), which is a key process for ATP synthesis. Glycogenolysis can be inhibited by elevated $[\text{H}^+]$ as well (Chasiotis 1983). Inhibition of glycolysis impairs the production of ATP by both non-oxidative and oxidative pathways. Since

the glycolytic pathway itself produces a positive net amount of ATP, impairment of this pathway directly reduces ATP synthesis. Inhibition of glycolysis also affects oxidative phosphorylation because the end product of glycolysis, pyruvate, is used as a substrate for the citric acid cycle. The lower rate of ATP synthesis may negatively affect exercise performance if ATP demand exceeds the capacity of the system to re-synthesize ATP from ADP + P_i. Elevated [H⁺] also impairs the function of ATPases that hydrolyze ATP into ADP + P_i. Most importantly, impairment of the myosin ATPase in the myosin heavy chain filaments will diminish the contractile function of these fibers. The fast and very-fast myosin ATPases found in type IIa and type IIX fibers specifically are highly susceptible to elevated [H⁺] (Keyser 2010). High [H⁺] also reduces the number of actin-myosin cross-bridges that are formed, leading to a decrease in force production (Keyser 2010). This impaired contractile function has been attributed to a decrease in the number of cross-bridges that transition from a loosely-bound state to a tightly-bound state (which is a high-force generating configuration) during the power stroke.

During times of high ATP demand, such as high intensity exercise, preserving the ATP-synthesizing capability of the muscle and decreasing the rate of ATP usage are paramount in order to prolong ability of the working muscles to repeatedly contract. In order to mitigate the effects of [H⁺] increase, buffering occurs, which allows the intramuscular pH to remain relatively constant, even during intense exercise. The loss of force production in muscles is not solely dependent upon intramuscular pH, however, as research has shown that pH levels in isolated muscle preparations well below that observed in local muscular fatigue produce only half that observed in local muscular fatigue (Renaud, Allard et al. 1986). In addition, the Na⁺/K⁺ ATPases found in the sarcolemma and the Ca²⁺ ATPases found in the sarcoplasmic reticulum

(SR) membrane will also have impaired function, slowing the rate of repolarization of the sarcolemma and delaying Ca^{2+} sequestering in the SR.

Accumulation of other metabolites such as ammonium and P_i likely contribute to the development of fatigue as well. Ammonium (NH_4^+) production during exercise occurs when adenosine monophosphate is deaminated by adenylate deaminase to inosine monophosphate (IMP) and NH_3 and also from the purine nucleotide cycle (PNC) (Mutch and Banister 1983). Ammonia (NH_3) can serve as a buffer for H^+ , production ammonium ions, which as a beneficial effect for maintaining pH. While ammonium ions positively induce phosphofructokinase (PFK, a key regulatory enzyme of glycolysis) and produce fumarate (an intermediate of the Krebs cycle) through the PNC (Sugden and Newsholme 1975), excess ammonium ion accumulation inhibits isocitrate dehydrogenase and pyruvate dehydrogenase (Roberts and Smith 1989). Since these enzymes are both involved in oxidative metabolism, the inhibition of them reduces the rate of oxidative metabolism. Thus, ammonium ion production during the early stages of exercise serves to enhance glycolysis and Krebs cycle activity. Excess ammonium ion production and accumulation however, has detrimental effects that may contribute to fatigue development.

Inorganic phosphates (P_i) are a product of ATP hydrolysis. Among the effects of increased $[\text{P}_i]$ are a positive regulation of glycolytic activity (Sugden and Newsholme 1975) and increased binding of P_i to myosin, which may decrease force output (Cooke and Pate 1985). Increased $[\text{P}_i]$ does not seem to affect the maximum muscle fiber shortening velocity, but does decrease peak isometric tension (Cooke, Franks et al. 1988). This is likely due to a decreased number of actin-myosin cross bridges in the strongly-bound state. In addition, there is evidence that P_i can bind to free Ca^{2+} and form the precipitate CaP_i , which decreases the amount of Ca^{2+}

that is available for excitation-contraction coupling (Fryer, Owen et al. 1995, Dahlstedt, Katz et al. 2001, Dutka, Cole et al. 2005, Amann and Calbet 2008).

Both proton accumulation and P_i accumulation occur during times of decreased oxygen transport to working muscles, while the accumulation of these metabolites is decreased when oxygen transport to working muscles is high. An increase in oxygen transport decreases the production of both H^+ and P_i due to shifts away from non-aerobic glycolytic metabolism (and therefore decrease lactate production) and phosphocreatine hydrolysis (which causes the accumulation of cytosolic P_i). The increase in oxygen delivery simultaneously aids in the removal of these (and other) metabolites, thereby decreasing their accumulation. During sustained high-intensity exercise longer than four minutes, cardiac output (\dot{Q}) and leg blood flow approach the peak values and further increases to \dot{Q} and leg blood flow are no longer possible, which reduces the ability of the cardiovascular system to compensate for decreased arterial oxygen content (C_{aO_2}). A fall in C_{aO_2} is commonly seen in both elite athletes and normal humans during sea level exercise (Amann and Calbet 2008). This fall in C_{aO_2} is generally due to a fall in the arterial partial pressure of oxygen (P_{aO_2}) and corresponding decrease in arterial hemoglobin saturation (S_{aO_2}). This phenomenon is referred to as exercise-induced arterial hypoxemia, or EIAH (Dempsey and Wagner 1999). While the severity of EIAH varies greatly, most humans demonstrate at least mild EIAH during sea-level exercise, with S_{aO_2} values of approximately 93-95%. While this reduction in S_{aO_2} is small, it has been shown that a fall in S_{aO_2} of $> 3.0\%$ has significant detrimental effects on VO_{2max} (Harms, McClaran et al. 2000), which lead to impaired endurance exercise performance.

Impaired contractile function by elevated $[P_i]$ and $[H^+]$ can be mitigated by enhancing the overall contractile function of the muscles, which can be achieved in the periphery without

additional input. The contractile function of the skeletal muscles is enhanced at the cellular level through several mechanisms. These regulatory strategies preserve the force-producing capabilities of exercising muscles even during exhaustive exercise, thus helping mitigate the development of peripheral fatigue. There is evidence that even during full force development, intracellular calcium concentration ($[Ca^{2+}]_i$) is submaximal (Westerblad and Allen 1991). Reducing the need for high $[Ca^{2+}]_i$ for full force development reduces the amount of Ca^{2+} flux needed to achieve maximal contraction. This also reduces the ATP demand since the re-sequestering of Ca^{2+} into the sarcoplasmic reticulum requires ATP hydrolysis. The ability of muscle fibers to generate full force even when $[Ca^{2+}]_i$ is submaximal is due to elevated Ca^{2+} sensitivity in the muscle fiber following activation.

An increase in $[Ca^{2+}]_i$, in addition to allowing muscle contraction to occur, also serves to increase Ca^{2+} sensitivity through myosin regulatory light chain (RLC) phosphorylation (MacIntosh, Grange et al. 1993). During muscle contraction when Ca^{2+} is released from the sarcoplasmic reticulum, it binds to troponin in order to move tropomyosin and expose actin binding sites to myosin. Ca^{2+} also binds to calmodulin, a small protein. The Ca^{2+} -calmodulin complex is able to bind to MCLK and activate it. The RLC is phosphorylated by activated myosin light chain kinase (MLCK). This increase in sensitivity has been shown to be due to an increase in the number of actin-myosin cross-bridges, which is accomplished by an increase in the rate of engagement of these cross-bridges, not a decrease in the rate of cross-bridge dissociation (Sweeney and Stull 1990, Levine, Kensler et al. 1996).

Increasing Ca^{2+} sensitivity preserves the ability of the muscle to contract and produce force while decreasing the amount of Ca^{2+} that needs to be released. This decrease in Ca^{2+} release is mediated by the Ca^{2+} -calmodulin complex, which inhibits Ca^{2+} release in addition to

activating MLCK. The reduction in Ca^{2+} release decreases the need for Ca^{2+} pumping, thus preserving ATP within the muscle cell (since ATP is needed to power Ca^{2+} pumps in membrane of the sarcoplasmic reticulum) and allowing for prolonged muscle activation during exercise (MacIntosh, Holash et al. 2012). A reduction in the activity of the Ca^{2+} ATPase can have significant consequences since it has been estimated that the Ca^{2+} ATPase contributes as much as 30% of total ATP use, with the Na^+/K^+ -ATPase contributing approximately 10% and the myosin ATPase ~60% (Homsher 1987). Of these ATPases, only the myosin ATPase uses ATP to generate force and do work. The other two ATPases are ion pumps which establish electrochemical gradients essential to the function of the muscle.

Ca^{2+} release can be affected by membrane excitability as well, since the release of Ca^{2+} is typically triggered by an action potential traveling down an α -motoneuron to a muscle fiber and propagating through transverse tubules deep into the muscle fiber (Song, Lee et al. 2011). The concentration of intracellular and extracellular ions and ion channel activity affect the magnitude of Ca^{2+} release, which can be attenuated during times of metabolic stress in order to preserve ATP (MacIntosh, Holash et al. 2012). The changes in membrane excitability at the onset of exercise are complex, but some factors involved are the increase in extracellular potassium ion concentration ($[\text{K}^+]_e$) (Mohr, Nordsborg et al. 2004, Nielsen, Mohr et al. 2004) and a concomitant decrease in the activity of voltage-dependent chloride channel proteins (ClC-1) (Pedersen, de Paoli et al. 2009). The reduction in membrane excitability via ClC-1 and ATP-sensitive K^+ channels (K_{ATP} channels) inhibits ryanodine receptor (RyR) mediated Ca^{2+} release.

Regulation of RyR is important because this controls Ca^{2+} release, which in turn affects ATP usage. During exercise the changes in the metabolic milieu of the muscle can elicit changes in RyR opening probability (P_o), thereby modulating RyR-mediated Ca^{2+} release. Changes in

cellular ATP, Ca^{2+} , and Mg^{2+} concentrations during exercise have direct effects on RyR P_o since all of these can serve as regulatory ligands of RyR (MacIntosh, Holash et al. 2012). In addition, reactive oxygen species (ROS), which are known to be produced during exercise (Davies, Quintanilha et al. 1982, Alessio, Hagerman et al. 2000), and modulate RyR-mediated Ca^{2+} release (Oba, Murayama et al. 2002).

RyR regulation by ATP, Ca^{2+} , and Mg^{2+} occurs via two binding sites. The A-site is an activation regulating site whereas the I-site is an inhibitory site, which is a lower affinity site (Lamb 2000). The concentration of Mg^{2+} at rest is sufficient to have an inhibitory effect on RyR, overriding the activating effect of both ATP and Ca^{2+} . Despite this strong inhibition of RyR at rest, an action potential can activate RyR via voltage-sensitive dihydropyridine receptors (DHPRs), which are directly connected to RyR and are capable of mechanically opening the channel when an action potential is detected (Tanabe, Beam et al. 1990, Lu, Xu et al. 1994).

During exercise, Mg^{2+} is released during ATP hydrolysis, since it is associated with both ATP and ADP, but has a lower affinity for ADP than ATP. The simultaneous increase in $[\text{Mg}^{2+}]$ and decrease in $[\text{ATP}]$ cause a decrease in RyR P_o , which attenuates Ca^{2+} release. Thus, the control of Ca^{2+} release is a key strategy for conserving ATP and is achieved by a diverse array of mechanisms working at various locations in the cell.

It is clear that there are many peripheral factors that play into the regulation of force production (and by extension, exercise intensity) during sustained or repeated muscle contractions. Figure 3 provides a summary of several peripheral factors thought to limit $\text{VO}_{2\text{max}}$ (and by extension, performance). There are many diverse peripheral strategies to mitigate the development of fatigue in the muscle milieu, which range from changes in contractile function to

buffering of organic acids. The biochemical changes in exercising muscle environment may have both direct and indirect effects on the development of fatigue.

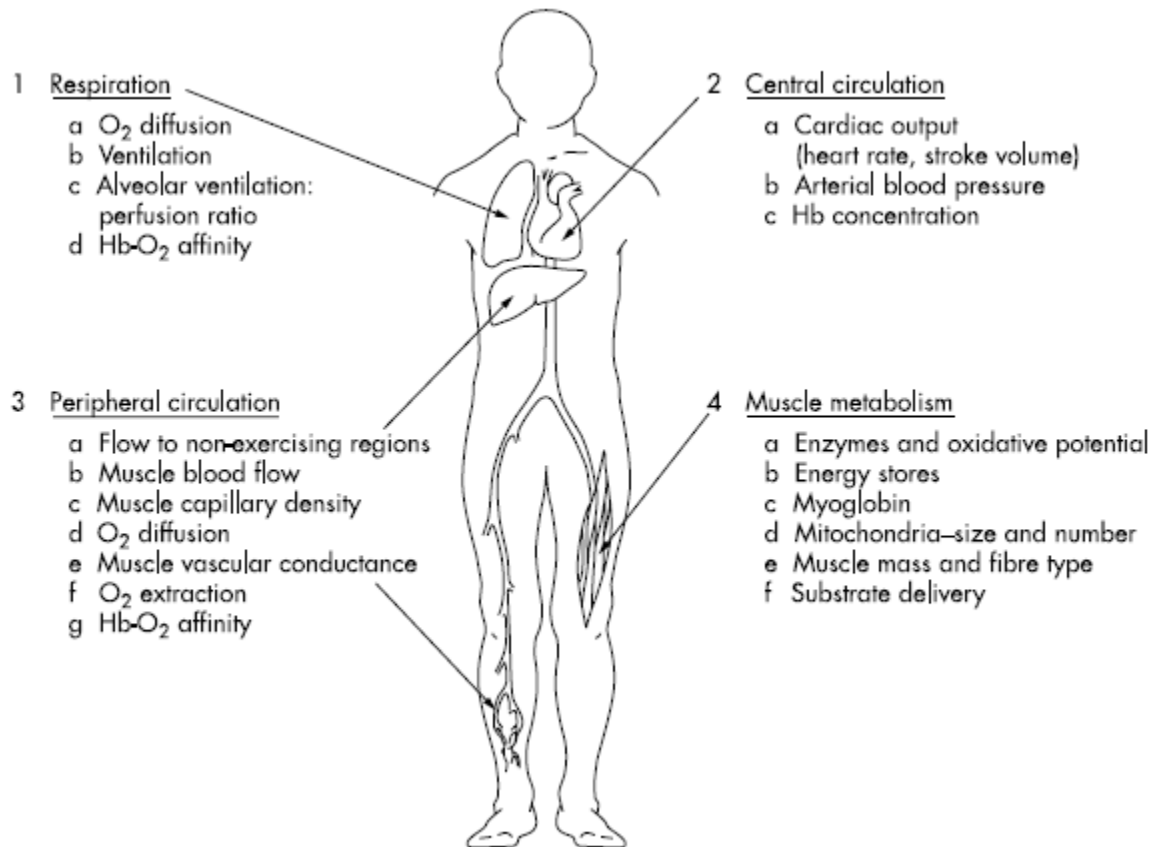


Figure 3. Classic diagram depicting limitations to $\text{VO}_{2\text{max}}$; note the absence of the central nervous system in the diagram. (Noakes and St Clair Gibson 2004)

Central Model

In the central model, instead of fatigue originating in the periphery (e.g. working muscles), fatigue is derived from central factors such as motivation, central nervous system transmission, or motor unit recruitment (Kirkendall 1990). Central fatigue occurs proximal to the neuromuscular junction (Bigland-Ritchie, Jones et al. 1978), therefore impairment of the α -motoneurons, spinal cord, or motor cortex may contribute to the development of central fatigue. Central fatigue is commonly assessed using maximal voluntary contractions with super-imposed

stimulation of either the motor cortex using transcranial magnetic stimulation (TMS) or motor-point stimulation by electrical stimulation of a peripheral nerve (Gandevia, Allen et al. 1996).

Supraspinal factors appear to influence the excitability of the cortex in fatigued humans (Taylor, Butler et al. 1996), which may contributed to altered muscle activation by the motor cortex. Inadequate neural drive upstream of the motor cortex may contribute to the development of central fatigue. The level of voluntary activation of α -motoneurons is influence by the afferent feedback of muscle afferents. Relaxed, un-fatigued human biceps brachii muscle can have greater than 99% voluntary activation whereas in the fatigued state voluntary activation drops to about 90% (Gandevia, Allen et al. 1996). This decrease in voluntary activation suggests that central fatigue developed after a 3 minute maximal effort. The authors attributed this sub-optimal voluntary activation to sub-optimal activation of the motor cortex. This study was not able to discern where in the central nervous system the central fatigue originated because of the limitations of using twitch interpolation by motor-point stimulation. The results from the Gandevia et al. study indicated that each part of the motor pathway between the motor cortex to the muscle fibers were not operating at their maximal limit even during maximal voluntary effort. Stimulation of the motor cortex via TMS produced additional force output from the biceps brachii, indicating that there was some degree of reserve for force generation.

Alterations in the brain itself may contribute to the development of central fatigue. It has been proposed that brain serotonin (5-hydroxytryptamine, or 5-HT) may alter exercise performance by promoting central fatigue development (Newsholme, Ackworth et al. 1987). 5-HT is a neurotransmitter that is known to affect arousal, lethargy, sleepiness, and mood (Davis 1995) and the synthesis and turnover of 5-HT may be increased during exercise. 5-HT is synthesized from tryptophan (TRP), which is transported into the brain at a higher rate during

exercise because of the increase in branched-chain amino acid (BCAA) uptake during exercise (Davis 1995). BCAAs are able to be oxidized to produce energy and therefore play an important role in maintaining energy supply during exercise, supplementary to carbohydrates and free fatty acids (FFA). Since BCAAs and TRP share a common transport mechanism into the brain, as more BCAAs are taken up into the brain, the ratio of TRP to BCAAs increases and thus more TRP is transported into the brain and synthesis of 5-HT increases. Concurrently, the increase in FFA oxidation during exercise increases FFA circulation in the blood. This displaces TRP from the carrier albumin and increases the amount of free (versus bound) TRP in the blood, which increases the ratio of TRP to BCAAs as well.

An increase in brain 5-HT seems to impair the function of the central nervous system during exercise, which in turn impairs exercise performance. 5-HT appears to have a suppressive effect on central motor drive (CMD), so as 5-HT levels in the brain accumulate, CMD is impaired. During prolonged exercise, 5-HT accumulates in the brain while the neurotransmitter dopamine (DA) also increases in the brain (Blomstrand, Perrett et al. 1989). DA is known to play a role in arousal, motivation, and neuromuscular control, but as fatigue develops, the activity of brain DA decreases (Davis 1995). Both the decrease of DA levels as fatigue develops and the concurrent increase in 5-HT levels appears to contribute to the development of central fatigue.

Administration of drugs that alter brain 5-HT has been shown to alter exercise performance (Davis 1995). Administration of a 5-HT receptor agonist has been shown to cause a decrease in time-to-exhaustion in exercising rats (Bailey, Davis et al. 1993) whereas administration of a 5-HT antagonist increased the time-to-exhaustion (Bailey, Davis et al. 1992). The altered brain activity as a result of these pharmacological interventions did not appear to

affect hormonal or substrate responses, suggesting that the alteration in fatigue development was due to changes in brain activity (Bailey, Davis et al. 1993). Such variables as body temperature, blood glucose, muscle and liver glycogen, and stress hormone levels did not appear to account for the change in fatigue development observed following pharmacological intervention with 5-HT agonists and antagonists. Further studies conducted on humans verified these results (Wilson and Maughan 1992, Davis, Bailey et al. 1993).

An increase in firing of afferent fibers from the muscles, joints and tendons may contribute to the development of central fatigue (Gandevia, Allen et al. 1996). This may decrease the drive to the corticospinal neurons, despite normal excitability in these neurons. Specifically, the activity of group III and IV muscle afferents may act at a supraspinal level and diminish motor output from the cortex (Martin, Weerakkody et al. 2008, Taylor and Gandevia 2008). These fibers sense changes in the metabolic milieu of exercising muscle fibers and project centrally, resulting in alterations in CMD (Gandevia 1998, Amann 2011).

Group III fibers are myelinated while group IV fibers are unmyelinated fibers, but both project to various sites throughout the central nervous system via the spinal cord (Amann, Blain et al. 2011), playing an important role in reflex responses of various systems including cardiovascular and respiratory reflexes. Pharmacologically blocking these afferent fibers compromises circulation and pulmonary ventilation during cycling exercise, which encourages the development of arterial hypoxemia and respiratory acidosis (Amann, Blain et al. 2011). Further research has shown that the activity of these group III and IV fibers impairs CMD (Amann, Blain et al. 2011), but also stimulates the proper ventilatory and circulatory responses to exercise. Blocking the spinal receptors of these afferent fibers resulted in an increase in CMD during cycling exercise, but also a greater development of peripheral fatigue due to decreased

circulation and ventilation. Thus it is clear that the group III and IV fibers link the regulation of central and peripheral fatigue. While this complicates the role of group III and IV afferents, it also illustrates that the feedback they provide is crucial for optimal endurance exercise performance. The impairment in CMD from group III and IV afferents hinders performance during endurance exercise (e.g. maximal cycling time trial), but the effects of this hindrance are overridden by the positive effects these fibers have on circulation and pulmonary ventilation; however, the impairment in CMD does not seem to be a permanent hindrance. It is commonly observed that in the final stages of a cycling time trial, subjects are able to increase their performance, overcoming this impairment in central motor drive (Amann, Eldridge et al. 2006, Amann and Dempsey 2008). This is commonly referred to as the “end-spurt” phenomenon (Noakes 2011).

The end-spurt cannot be explained by either the central or peripheral models. It is unclear what enables subjects who are presumably fatigued from exercising at maximal effort for the majority of a self-paced time trial to improve their performance at the end of the time trial. The end-spurt phenomenon suggests that there is conscious input from the sensory organs and brain to the regulation of central motor drive (Noakes 2007). Spinal and supraspinal factors alone fail to explain why the central motor drive can be increased at the end of a time trial, nor do they explain how it is possible for a subject to know where the end of the time trial is without conscious input. Therefore an updated model has been proposed that may explain why the end-spurt phenomenon can occur (Noakes 1997).

Central Governor Model

The central governor model is a relatively new model which proposes that there is a regulatory mechanism in the subconscious brain that integrates conscious thought, afferent

signals, and other information and continuously modifies central motor drive in order to maintain homeostasis (Noakes, St Clair Gibson et al. 2004, Noakes, St Clair Gibson et al. 2005). Before exercise even begins, the so-called “central governor” selects an optimal pacing strategy that will preserve internal homeostasis in a process that has been dubbed “teleoanticipation” (St Clair Gibson and Noakes 2004). During exercise this pacing strategy is continuously adjusted in response to various inputs to the central governor.

The optimal pacing strategy selected by the central governor maximizes performance without perturbing whole-body homeostasis. This is achieved by setting a relative maximal tolerance for exercise intensity below the absolute maximum. Evidence of the existence of a “reserve” capacity is emerging and several studies have shown that by manipulating subjects using false competition (either simulated head-to-head competition or false representation of a prior performance by that individual), an improvement in time-trial performance can be elicited (Corbett, Barwood et al. 2012, Stone, Thomas et al. 2012). Further evidence exists that complete muscle activation during exercise does not appear to occur (Sloniger, Cureton et al. 1997, Sloniger, Cureton et al. 1997, Amann, Eldridge et al. 2006, Amann, Romer et al. 2007), which may serve as a protective mechanism. Submaximal ($< 100\%$ activation) recruitment of muscle fibers reduces the peripheral demand for \dot{Q} and preserves a larger portion of \dot{Q} which can be delivered to the heart and respiratory muscles.

This model of regulations does allow for the existence of an end-spurt because conscious thought is an input to the central governor, which can then use that information to modify CMD accordingly. Furthermore, previous experience also exerts an influence on the anticipatory component of the pacing strategy that is selected prior to exercise (Tucker 2009), and so a reserve may be held in order to facilitate the end-spurt at the end of exercise. Throughout the

course of exercise, the “template” that is adopted by the central governor provides a reference as the central governor receives feedback and adjustments are continually made to optimize performance. As the subject becomes aware of the impending end of the exercise bout, the CMD becomes less limited and an increase in power output is observed. This could be in part due to an increase in motivation, but may also be due to the afferent input signaling the presence of adequate bioenergetic resources and a stable enough metabolic milieu to allow an increase in CMD without disrupting homeostasis. Figure 4 illustrates the teleoanticipatory model of the regulation of exercise.

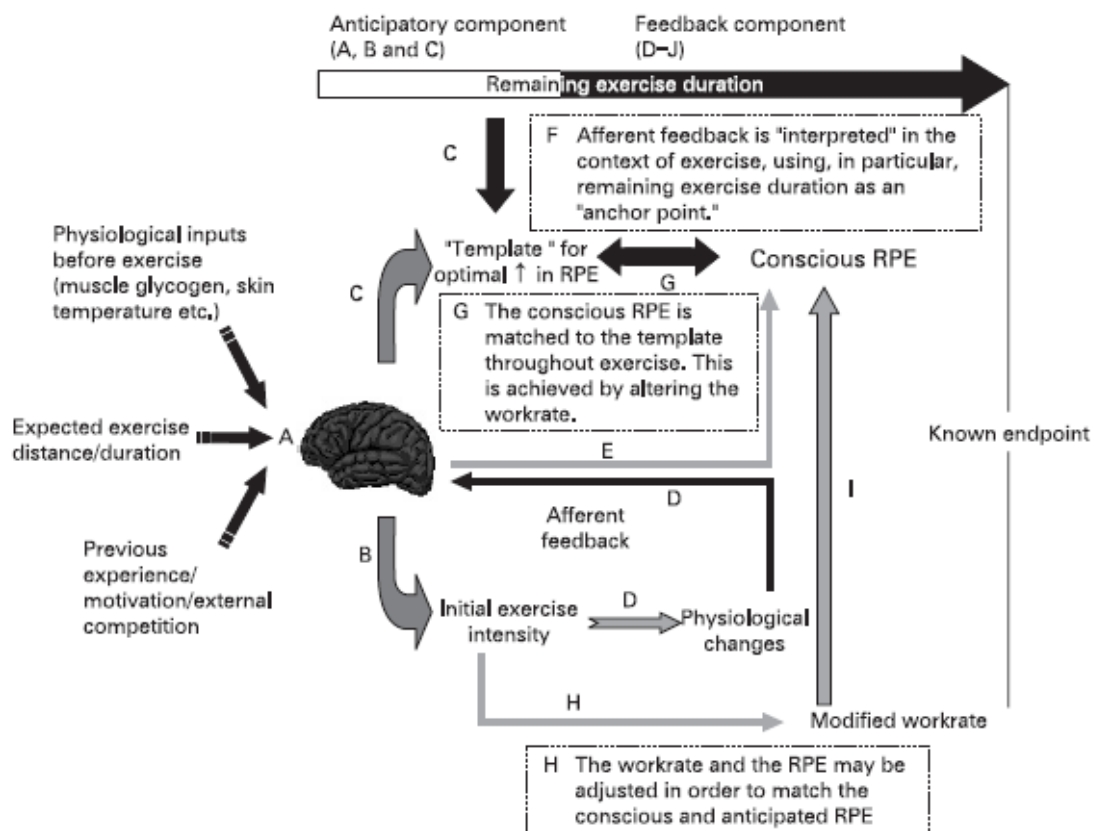


Figure 4. Model for anticipatory regulation of exercise during self-paced exercise. Black shading denotes input to the brain while gray shading denotes output or efferent processes. (Tucker 2009)

While whole-body afferent information is relayed to the central governor, certain inputs almost certainly exert a more significant influence than others on efferent neural output. For example, the metabolic status of smaller muscle groups not commonly used heavily in endurance exercise like the extensor digitorum longus may not be as important as the metabolic status of the heart. The threat of ischemia to the heart would provide an overwhelmingly strong input to the central governor and the corresponding modification of CMD would serve to preserve cardiac blood flow in order to avoid ischemic conditions on the myocardium (Noakes, Peltonen et al. 2001). The peripheral model suggests that hypoxic conditions in the muscles and heart result in an increase in \dot{Q} , whereas the central governor model predicts that hypoxic conditions in the muscles and heart result in a proportionally lower \dot{Q} in order to prevent damage to the heart (Noakes, Peltonen et al. 2001). This protective mechanism also ensures that the brain and respiratory system receive adequate blood flow as well, which prevents catastrophic homeostatic failure. The output from the central governor will also decrease limb locomotor drive and thus reduce the demand of the exercising muscles for blood flow, allowing a larger proportion of the reduced \dot{Q} to be sent to the vital organs.

Pacing Strategy

Selecting an optimal pacing strategy during exercise is critical when an athlete is seeking maximum performance. An optimal pacing strategy allows an athlete to manage their bioenergetic stores (e.g. muscle and liver glycogen), prevent substrate depletion, and minimize metabolite accumulation during exercise (Foster, Schrager et al. 1994, Tucker and Noakes 2009). The pacing strategy that is adopted at the onset of exercise has been shown to be dependent on

many factors including the expected duration and previous experience (Tucker 2009, Noakes 2012). This is particularly evident when novice and experienced cyclists are compared.

Experienced cyclists tend to have imbedded pacing schemas for tasks with which they are familiar (Mauger, Jones et al. 2010). In addition, these pacing schemas are robust and uninfluenced by variations in the pacing strategy in the same exercise task (Mauger, Jones et al. 2010). In addition, experienced cyclists who are accustomed to receiving feedback on their status throughout an exercise task (e.g. distance knowledge and distance feedback), can use this information to optimize their pacing strategy throughout a time trial and therefore improve performance whereas novice cyclists do not exhibit this improvement with proper feedback (Williams, Bailey et al. 2012). It follows that familiarization with a task and training status seem to play a key role in the development of an optimal, robust pacing schema for a given exercise task (Noakes 2012).

Deception

Deception has long been a topic of interest for athletic performance. There is substantial evidence that deception can alter athletic performance (Ness and Patton 1979, Ansley, Robson et al. 2004, Paterson and Marino 2004, Morton 2009, Micklewright, Papadopoulou et al. 2010, Billaut, Bishop et al. 2011, Corbett, Barwood et al. 2012, Stone, Thomas et al. 2012). The cause of this alteration in athletic performance remains unclear; however it appears that both psychological and physiological factors play a role.

In a study conducted by Ness and Patton, college-aged males (48 college males) performed a single, maximal inclined bench press exercise once a week for 6 weeks. After three weeks, the subjects were deceived by altering the resistance. The researchers re-labeled the weights with either higher or lower numbers and in one condition removed the labels altogether.

Therefore, the true resistance was increased in one condition, decreased in a second condition compared to what the subjects believed they were lifting, and knowledge of resistance was withheld in a third condition. Subjects had significantly higher strength performance when resistance was greater than subjects believed. The authors attributed performance difference was attributed to the subjects' attempts to remain consistent with self-expectations.

Further research on deception conducted by Ansley et al. in 2004 revealed that the anticipated exercise duration and previous experience with an exercise task sets a pre-programmed end point (Ansley, Robson et al. 2004). In this study, the authors recruited eight physically active males who then performed six Wingate Anaerobic Tests (WAnT) on a cycle ergometer. The investigators did not report whether or not these subjects were trained cyclists; however the subjects used cleated cycling shoes, suggesting that they had some degree of cycling experience. The subjects in this study were told that they were performing four, 30-second WAnT, one 33-s WAnT, and one 36-s WAnT throughout the course of the study. The standard WAnT is a 30-second test, so the 33-s and 36-s conditions were longer than normal (by 3- and 6-seconds, respectively). The investigators in fact had the subjects perform two WAnT of each duration (30-s, 33-s, and 36-s) without telling the subjects. Therefore in one 33-s trial and one 36-s trial, subjects actually believed that they were completing a 30-s WAnT when they were actually completing a longer WAnT. The power output at the end of the 36-s deception trial was lower than in the 36-s informed trial, as was the mean power output. There was no difference, however, for mean power during the 33-s and 36-s trials. The power during the first 30-s of all trials was not significantly different, regardless of the final duration of the trial. The authors concluded that there is a “pre-programmed 30-s end point” for subjects completing a WAnT and that the end point is set by previous experience and anticipated exercise duration. In addition,

they suggest that power output is controlled by factors other than the capacity of skeletal muscle energy reserves and maximal rate of substrate utilization.

Another study investigating the effect of deceptively altering the distance of a time trial found that subjects perform subsequent trials based on perceived effort rather than the actual distance (Paterson and Marino 2004). In this study 21 endurance-trained cyclists participating, completing three self-paced time trials. The first and third time trials were 30-km in length. The second trial was either 30-km, 24-km (deceptively short), or 36-km (deceptively long), but all subjects were told that the trial was 30-km in length, regardless of which group they were in. The time to completion for the third time trial was decreased for the deceptively long group, increased in the deceptively short group, and unchanged in the control group. The authors concluded that the data supports the subconscious control of efferent neural command and that perceived effort played a more significant role in determining the performance of trained cyclists rather than the actual distance of a time trial. It is interesting to note that there was an end-spurt observed in all conditions.

Yet another study on deception conducted by Morton in 2009 found that subjects were able to increase their cycling time to exhaustion when a clock visible to the subjects was calibrated to run slower than normal (Morton 2009). Subjects in this study were 6 male and 6 female regularly training and playing members of a university soccer club who did not compete in any other sports. The subjects completed three rides to exhaustion on a cycle ergometer. During these trials they were allowed to read the elapsed time on a large digital clock. The investigators manipulated the calibration of the clock in three separate ways. In one condition the calibration was accurate whereas in the other two conditions the clock was calibrated either 10% faster or 10% slower than normal. The tests were conducted in a double-blind and fully

counterbalanced (within genders) manner. All subjects were unaware of the deceptive changes in clock calibration. When the clock ran slow the subjects were able to cycle for a longer time before exhaustion compared to normal. Interestingly, there was no corresponding negative effect observed when the clock ran fast. The authors speculated that the improvement in performance was a result of increased motivation to try and remain consistent with self-expectations. This is consistent with the findings of Ness and Patton. The subjects in this study were not trained cyclists, which may limit the applicability of these results to cyclists. The main factor for the improvement of these subjects appeared to be increased motivation to remain consistent with self-expectations, a trait which competitive cyclists would surely possess.

A study conducted by Corbett et al. found that simulated head-to-head (HH) competition elicited performance improvements (Corbett, Barwood et al. 2012). A drawback of this study is that the participants were not trained cyclists. They were males who regularly participated in physical exercise and were accustomed to maximal exercise, but not specifically cycling. Subjects performed several 2,000-m cycling TTs, including three familiarization/baseline trials. The two experimental trials consisted of a solo TT on the same course and a TT with simulated HH competition. Subjects were told in the HH condition that they were competing against another individual of similar ability when in fact this was a sham. The on-screen avatar was in fact the subjects' own best baseline performance and the "competitor" was an investigator cycling at a moderate intensity on the other side of a translucent screen. This deception elicited improved performance from the subjects and the improvement was due to an increase in anaerobic energy yield. This supports the existence of a metabolic reserve which can be accessed through deception. The results of this study are consistent with the results of Ness and Patton since subjects were likely attempting to remain consistent with self-expectations of being

able to best a competitor of similar ability. The presence of competition may elicit increased motivation or have a dissociative effect which exerts a central influence and allows a greater utilization of the reserve capacity of an athlete (Corbett, Barwood et al. 2012).

The deception study conducted by Stone et al. studied trained male cyclists, investigating whether or not performance in a 4-km cycling TT could be improved by misleading the participants into believing that they were racing against their own best trials when in fact they were racing an avatar set at 102% of their baseline average power output (Stone, Thomas et al. 2012). Following a familiarization and baseline, subjects performed two experimental trials in which they competed against an on-screen avatar representing either their true baseline (ACC condition) or 102% of their baseline (DEC condition). Regardless of whether the true condition was ACC or DEC, subjects were informed that they were competing against their own baseline performance. The trials were randomized and counter-balanced.

In the DEC condition, time to completion was significantly better compared to ACC and average power output was higher. The pacing strategy adopted by these subjects was altered as well, with an increase in anaerobic power contribution (P_{an}) in the DEC condition. The authors attributed the improved performance to this increase in P_{an} . This increase in P_{an} most pronounced in the final 10% of the time trial, suggesting that the end-spurt was larger in the deception condition. This release of inhibition of CMD during the final stages may be due to increased motivation and a corresponding change in the output of the central governor. The allowance of increased drive during the end-spurt raises the relative maximum that is allowed by the central governor and thus promotes access to the proposed metabolic reserve.

Interestingly, the subjects had a higher RPE in the DEC condition, suggesting that they were consciously exerting more effort compared to baseline despite the fact that they believed

that they were competing against their own baseline performance. It was unclear whether this competition alone elicited the performance improvement, but a subsequent study by the same group revealed that competition alone did not influence performance (Stone, Thomas et al. 2012).

CHAPTER THREE

METHODS

Subjects

Fourteen trained competitive male cyclists between the ages of 18-30 years old were recruited for this study. Subjects were non-smokers with no prior history of pulmonary or cardiovascular disease as assessed by questionnaire. All subjects regularly participated in a cycling training regimen and had at least one year of competitive cycling experience. Peak oxygen uptake was assessed with a graded exercise test and subjects meeting the inclusion criteria of a $\dot{V}O_{2\max} > 60 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ were admitted to the study. All subjects were asked to continue their normal diet and regular training regimen throughout the duration of the study. Subjects were asked to refrain from strenuous exercise for 24 hours prior to each testing session and also to avoid caffeine and alcohol during this time period. All testing procedures and the informed consent were approved by the Indiana University Human Subjects Committee. Written informed consent was obtained before the subjects enrolled in the study.

Study Design and Protocol

All subjects completed four simulated 4-kilometer time trials (TT) on an electromagnetically braked cycle ergometer (Velotron Racermate, Seattle, WA, USA). Time to completion and power output were recorded to assess performance. Pacing strategy over the course of each TT was evaluated for all trials. Initially, subjects were informed that the purpose of the study will be to assess the consistency of 4-km cycling time trial performance when racing against their previous performance. Subjects were informed of the true nature of the study upon completion of the final experimental trial. In the first experimental trial, subjects completed a 4-km familiarization trial in order to ensure that the novelty of the TT and equipment did not

influence performance and to minimize the learning effect. The second experimental trial was a baseline assessment of the subjects' maximal effort for a 4-km TT. The third experimental trial was a deception trial in which subjects competed against an avatar representing 102% of their baseline average power output. Subjects were falsely informed that the avatar represented their own baseline performance. The fourth experimental trial was a knowledge of deception trial in which subjects competed against an avatar representing 102% of their baseline average power output. In this condition, subjects were aware of the true nature of the avatar and of the previous deception. Testing of each individual subject was conducted at the same time of day to minimize circadian variation. Pilot data collected ($n = 8$) prior to the start of data collection for this study observed no significant order effect between five repeated 4-km cycling time trials with no intervention ($p > 0.05$ between all trials).

Graded Exercise Protocol (Cycle Ergometer)

Subjects came to the laboratory and rested for several minutes before commencing exercise. The exercise testing was completed on an electromagnetically braked cycle ergometer (Velotron Racermate, Seattle, WA, USA). The seat height was adjusted to the subject's preferred position prior to the cycling test and the height was measured and recorded so it could be replicated for subsequent cycling tests (i.e. the time trials). Subjects were allowed to bring in their own pedals and shoes (e.g. clipless cycling pedals commonly used by cyclists) as well as their own saddle (bike seat) if desired. Resting measurements were collected for 3-5 minutes. Immediately after the resting period, subjects began to cycle at a workload of 100 Watts (W) and the workload was increased by 25W every minute until power output was not able to be maintained by the subjects (i.e. cadence drops by more than 20 rpm) or the subject voluntarily ended the test (Duke, Stickford et al. 2013, Weavil, Stickford et al. 2013). Subjects were

allowed to choose their own preferred cadence for the test and the computer software automatically adjusted the electromagnetic brake to maintain the appropriate workload.

$\dot{V}O_{2\max}$ tests were considered valid if they meet two of the following criteria: 1) a heart rate $\geq 90\%$ of the age-predicted maximal heart rate ($220 - \text{subject's age}$); 2) a respiratory exchange ratio (RER) ≥ 1.10 ; and 3) evidence of a plateau in $\dot{V}O_2$ with an increase in exercise intensity, which is defined as ≤ 150 mL increase in $\dot{V}O_2$ with an increase in workload (Howley, Bassett et al. 1995).

Time Trials (Cycle Ergometer)

Subjects completed a total of four (4) separate self-paced, maximal-effort, simulated 4-kilometer time trials on the same electromagnetically braked cycle ergometer (Velotron Racermate, Seattle, WA, USA) interfaced with computer software (Velotron 3D, Racermate, Seattle, WA, USA). The computer software allows the experimenter to generate a course of a specific length and elevation change. In this case, a flat (0% grade), 4-kilometer course was used. The computer software also allows the instantaneous generation of an onscreen avatar, which illustrates the subjects' progress as they undertake the time trial. Avatars can be saved and replayed in subsequent trials, allowing for head-to-head competition. Each subject was allowed to bring in their own pedals and shoes as well as their own saddle if desired. The seat height from the $\dot{V}O_{2\max}$ test was replicated. Subjects completed a standardized warm-up consisting of 5 minute of cycling at a workload of 150 W followed by a subsequent 5 minutes of cycling at 70% of their power at $\dot{V}O_{2\max}$. Subjects were then instructed to complete the 4-kilometer time trial in the shortest time possible. Subjects were blinded to all feedback except distance covered. Standardized verbal encouragement was given at the end of each kilometer and subjects were asked to give ratings of perceived exertion (RPE) at the end of each kilometer.

The first time trial was a familiarization trial (FAM) in order to mitigate the learning effect and to ensure subjects were used to the equipment and procedures. The second time trial was a baseline (BAS) trial. The third time trial was a deception time trial (DEC) in which the subjects competed head-to-head against a computer-generated avatar representing 102% of their baseline average power output. The subjects were falsely informed that the avatar represented their own baseline performance. The fourth time trial was a knowledge of deception (KDE) trial in which subjects competed head-to-head against an avatar set at 102% of their baseline average power output. In the KDE trial subjects were informed of the true nature of the previous trial, and in addition they were informed that the avatar was still set at 102% of their baseline average power output. Subjects were informed of the prior deception one day prior to coming in for the final trial using a standardized debriefing script.

Research has shown that time trial protocols have higher reproducibility than time-to-exhaustion protocols (Jeukendrup, Saris et al. 1996). Indeed Jeukendrup et al. demonstrated that cycling at 75% of maximal power output until exhaustion had poor reproducibility while time trial cycling demonstrated the least amount of variation between trials. Due to the high fidelity with which subjects can reproduce time trial performance, time trial protocols may be more reliable indicators of performance than time-to-exhaustion protocols. Laboratory simulated 4-kilometer cycling time trials in particular have been investigated and shown to be a reliable measure of both performance and pacing strategy (Stone, Thomas et al. 2011).

Ventilatory and Metabolic Parameters

Metabolic and ventilatory variables were continuously monitored during all testing via open-circuit, indirect calorimetry. Subjects breathed into a thermoplastic face-mask (Hans Rudolph, Kansas City, KS, USA) which was attached to a one-way, non-rebreathing valve.

Expired minute ventilation (V_E) was calculated from the computer integration of the inspiratory flow signal using a pneumotachometer (Hans Rudolph, Kansas City, KS, USA). Expired gases were sampled from a 5-L mixing chamber at a rate of 300 ml/min and analyzed for O_2 and CO_2 fractions using rapidly-responding gas analyzers (Applied Electrochemistry, Pittsburgh, PA). The gas analyzers and pneumotachometer were calibrated prior to data collection using certified standard gases and a spirometer (Tissot, Switzerland). Data collection software (DasyLab, Measurement Computing, Norton, MA) was used to record the data and calculate RER, VCO_2 and $\dot{V}O_2$.

Pacing

Power data was recorded using computer software interfaced with the cycle ergometer (Velotron 3D, Racermate, Seattle, WA, USA). Average power output was calculated over 0.5-km segments for each time trial (for a total of eight, 0.5-km segments for each trial) in all conditions, for all subjects. The average power output over each 0.5-km segment served as an indication of the pacing strategy adopted by the subjects for a given trial.

Self-Motivation

Subjects' self-motivation was assessed using the previously validated Self-Motivation Inventory (SMI), which is a 40-item questionnaire which assesses self-motivation (Dishman, Ickes et al. 1980, Dishman and Ickes 1981, Heiby, Onorato et al. 1987, Raglin, Morgan et al. 1990). The questionnaire was administered to the subjects upon completion of the final experimental trial.

Data Analysis

Statistical analysis was conducted using SPSS 20.0 (IBM Corporation, Chicago, IL, USA) statistical software. Since the hypothesis that the KDE condition would produce

improvements following deception was dependent on subjects improving in the DEC condition, subjects who failed to improve their performance in the DEC trial were separated from those who improved their performance in the DEC trial and analyzed separately. Data was assessed for normality using the Shapiro-Wilk test. Mean power output and TTC for each trial condition were analyzed for differences using a one-way, within-factors analysis of variance (ANOVA) with repeated measures. Pacing data was analyzed for differences using a fully factorial 3 x 8 (trial condition vs. distance covered) repeated measures ANOVA. Sphericity of data was assessed using Mauchly's sphericity test. If sphericity was violated, the departure from sphericity (ϵ) was calculated. If ϵ was < 0.75 , the Huynh-Feldt correction factor (Huynh and Feldt 1976) was applied and if ϵ was > 0.75 , the Greenhouse-Geisser correction factor (Greenhouse and Geisser 1959) was applied. *Post-hoc* comparisons for significant main effects were made using the Bonferroni correction for multiple comparisons (Dunn 1961). Significance was accepted at $p < 0.05$. An *a priori* power analysis (G*Power 3.1.3, Franz Faul, Germany) revealed that fourteen subjects would be required to observe significant differences with a power of 0.95 ($\omega^2 = .2275$) (Stone, Thomas et al. 2012).

CHAPTER FOUR

RESULTS

The main aim of the study was to evaluate whether or not the improvement in performance and changes in pacing strategy continue after subjects are informed of deception during a previous cycling time trial. Data is presented as mean \pm standard deviation. The reliability of estimates is given as a 95% confidence interval.

Subjects

Fourteen male trained, competitive cyclists participated in the study out of a total of twenty-six competitive male cyclist volunteers who were recruited for this study. Twelve cyclists were excluded for failing to meet the inclusion criteria for $\dot{V}O_{2\max}$. All subjects were healthy, non-smokers, and listed no history of cardiovascular or pulmonary disease on the preliminary questionnaire. Mean age was 20.7 ± 1.0 years; mean stature was 179.1 ± 2.7 cm and mean weight 71.4 ± 4.8 kg. All subjects were competitive cyclists who had participated in a competitive cycling event within one calendar year of the study period and who were actively engaged in a competitive cycling training regimen, with a mean training history of 2.3 ± 1.8 years. All subjects enrolled in the study completed a valid $\dot{V}O_{2\max}$ test as assessed by the criteria described previously (see Ch. 3). Mean $\dot{V}O_{2\max}$ was 61.6 ± 2.3 mL \cdot kg $^{-1}\cdot$ min $^{-1}$. Subject characteristics are summarized in Table 1.

Subject	Height (cm)	Weight (kg)	Age (years)	Years Training	VO2max (L/min)	VO2max (mL/kg/min)
Mean	179.1	71.4	20.7	2.3	4.4	61.6
SD	2.7	7.8	1.0	1.8	0.3	2.3

Table 1. Subject characteristics.

Power Output

Significant differences were observed between trials in mean power output for subjects who improved in the DEC trial ($F = 15.88, p = 0.002$). Mean power output for subjects who improved in the DEC trial ($n = 10$) was 321.7 ± 25.8 W for the BAS trial, 339.4 ± 21.0 W for the DEC trial, and 341.2 ± 19.0 W for the KDE trial. Mean power output for each experimental trial for subjects who improved in the DEC trial is given in Figure 5. Subjects who did not improve in the DEC trial ($n = 4$) also demonstrated differences in mean power output between trials ($F = 18.89, p = 0.003$). Mean power output for these subjects was 335.2 ± 50.1 W in the BAS trial, 320.9 ± 55.2 W in the DEC trial, and 348.3 ± 60.0 W in the KDE trial.

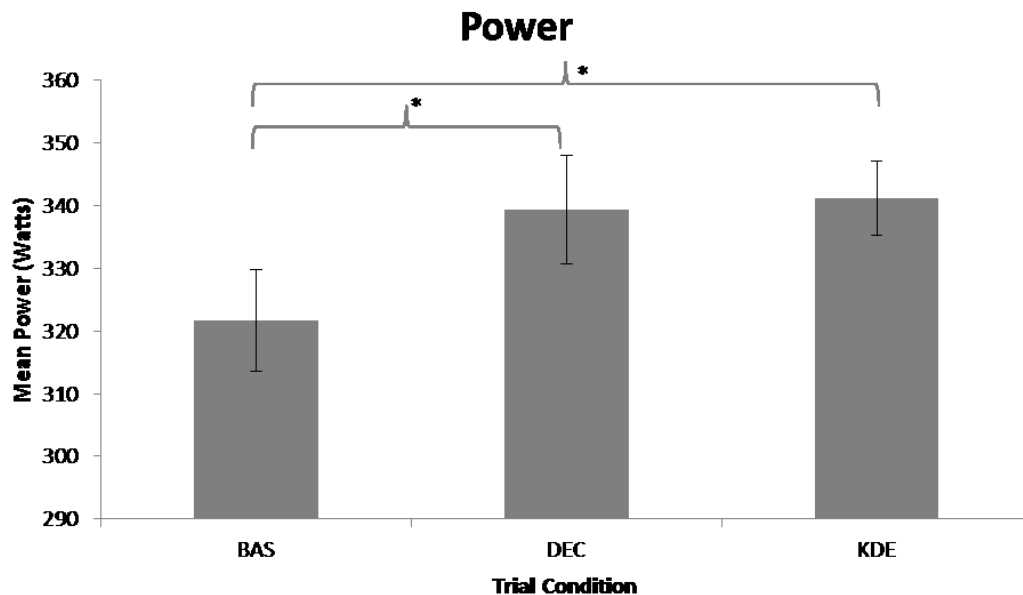


Figure 5. Mean power output in each experimental trial condition for subjects who improved from the BAS to DEC trials. Significant differences from BAS are denoted by an asterisk (* $p < 0.05$).

Pairwise comparisons revealed that mean power for the BAS trial in subjects who improved in the DEC trial was significantly lower than for the DEC and KDE trials ($p < 0.001$, 95% CI = -25.3 to -10.0, and $p = 0.014$, 95% CI = -34.8 to -4.1 W, respectively), but no significant differences were observed between the DEC and KDE trials ($p = 1.0$, 95% CI = -10.9 to 7.3 W). Pairwise comparisons for subjects who did not improve in the DEC trial revealed that mean power for the BAS trial was significantly higher than for the DEC trial ($p = 0.047$, 95% CI = 0.2 to 28.4 W), but not for the KDE trial ($p = 0.323$, 95% CI = -40.9 to 14.8 W). For these subjects, mean power in the DEC trial was significantly lower than the KDE trial ($p = 0.023$, 95% CI = -48.2 to -6.6 W). Mean power output in each experimental trial for subjects who did not improve from the BAS to DEC trials is given in Figure 6.

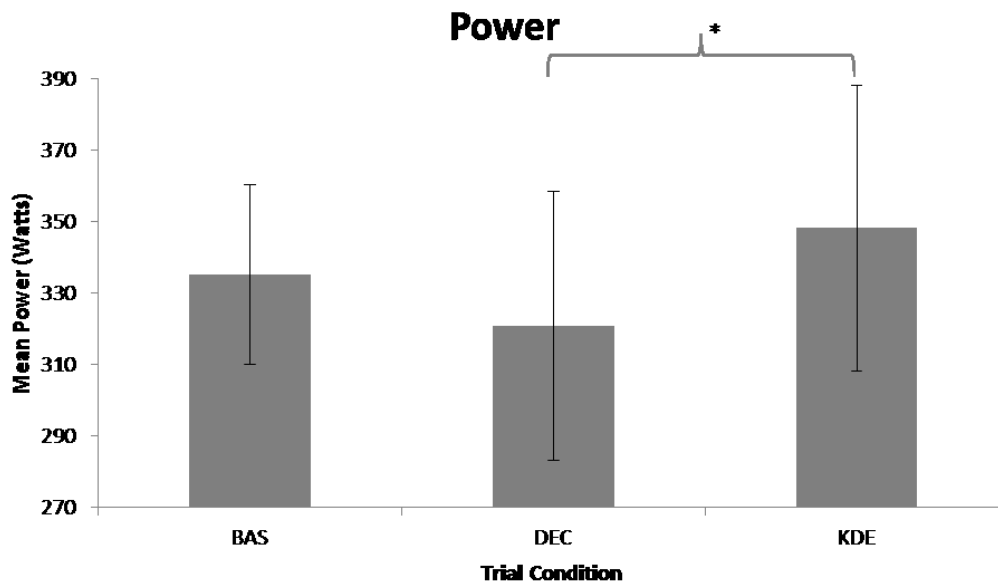


Figure 6. Mean power output in each experimental trial for subjects who did not improve from the BAS to DEC trials. Significant differences from DEC are denoted by an asterisk (* $p < 0.05$).

Time to Completion

Differences in TTC were observed between trials ($F = 13.61, p = 0.003$) for subjects who improved in the DEC trial ($n = 10$). For this group, mean TTC was 366.40 ± 11.38 s in the BAS trial, 358.61 ± 8.54 s in the DEC trial, and 358.12 ± 8.90 s in the KDE trial. Subjects who did not improve in the DEC trial ($n = 4$) also demonstrated differences in TTC between trials ($F = 17.31, p = 0.003$) and had mean TTCs of 361.12 ± 19.95 s in the BAS trial, 368.12 ± 24.25 s in the DEC trial, and 356.75 ± 23.70 s in the KDE trial. Figures 7 and 8 depict time to completion for individual subjects each respective group across all trials.

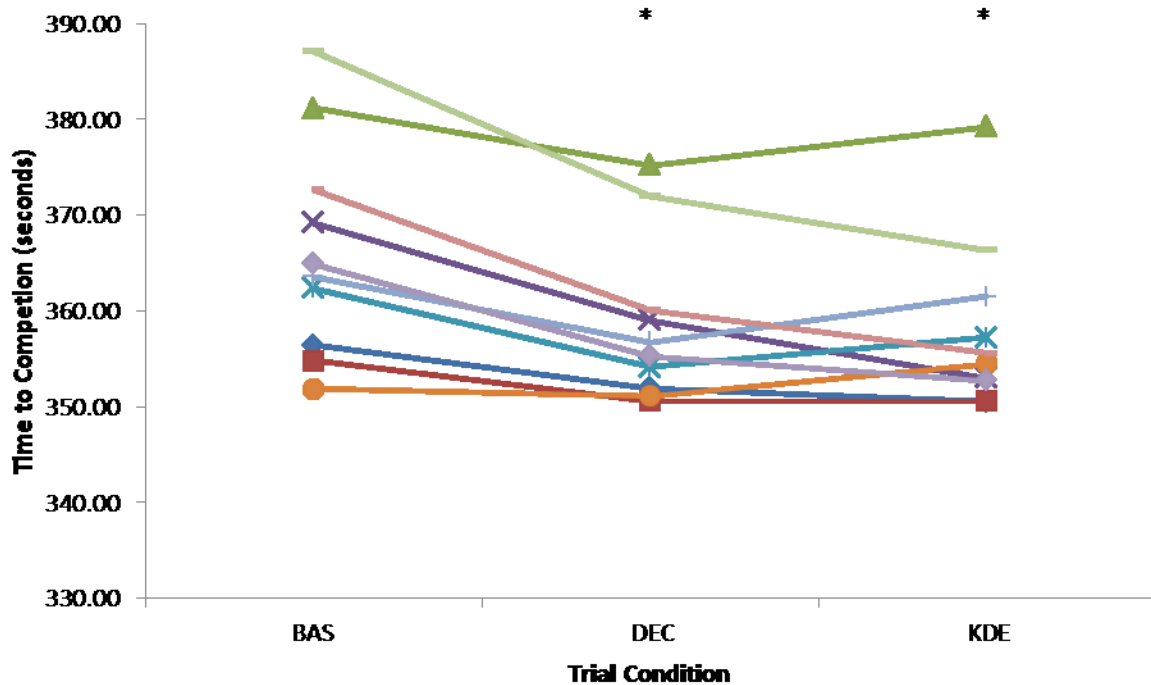


Figure 7. Time to completion for all trials for individual subjects who improved from the BAS to the DEC trials. Significant reductions in time to completion from BAS are denoted by an asterisk (* $p < 0.05$).

Pairwise comparisons revealed that TTC was significantly shorter in the DEC and KDE trials compared to the BAS trial in subjects who improved in the DEC trial ($p = 0.001$, 95% CI = -11.8 to -3.8 s and $p = 0.024$, 95% CI = -15.5 to -1.1 s). However, no difference between the DEC

and KDE trials was observed in these subjects ($p = 1.0$, 95% CI = -3.3 to 4.3s). For subjects who did not improve in the DEC trial, pairwise comparisons showed no difference between TTC in the BAS trial compared to the DEC trial ($p = 0.157$, 95% CI = -17.9 to 3.9 s) and the BAS trial compared to the KDE trial ($p = .421$, 95% CI = -6.3 to 15.0 s).

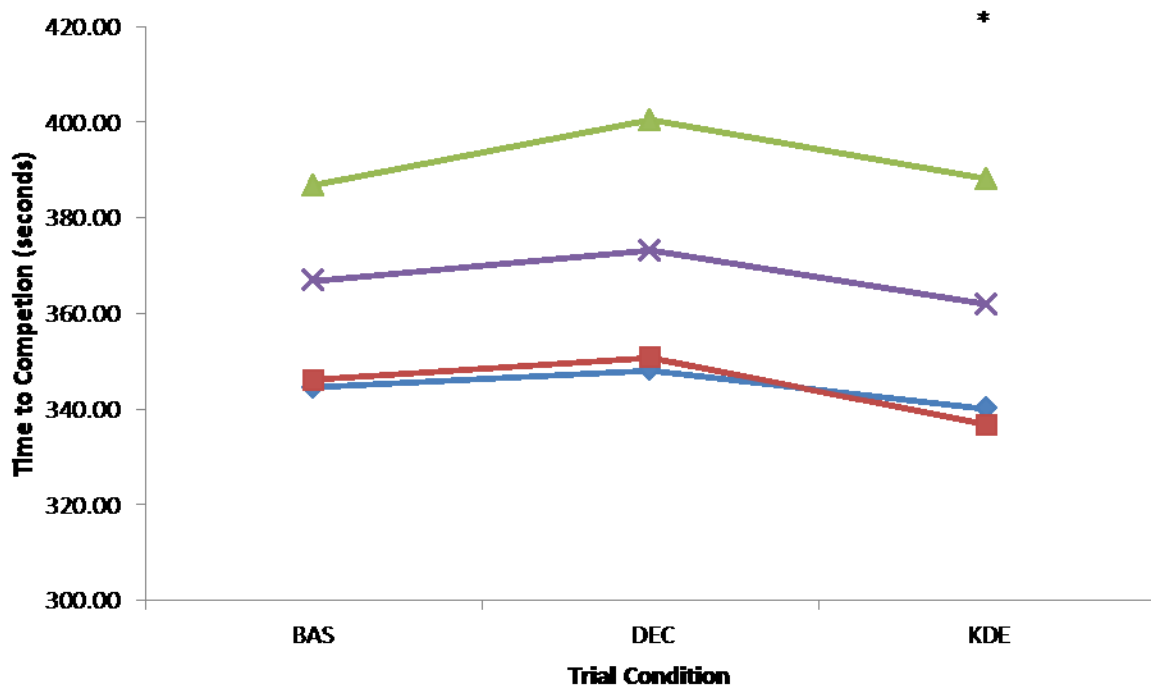


Figure 8. Time to completion for all trials for individual subjects who did not improve from the BAS to the DEC trials. Significant reductions in time to completion from DEC are denoted by an asterisk (* $p < 0.05$).

However, TTC in the DEC trial was significantly higher compared to the KDE trial ($p = .009$, 95% CI = 5.3 to 17.5 s). Figure 9 displays the mean time to completion for all trials for only subjects who improved from the BAS to the DEC trial. Figure 10 displays the mean time to completion for all trials for subjects who did not improve from BAS to DEC.

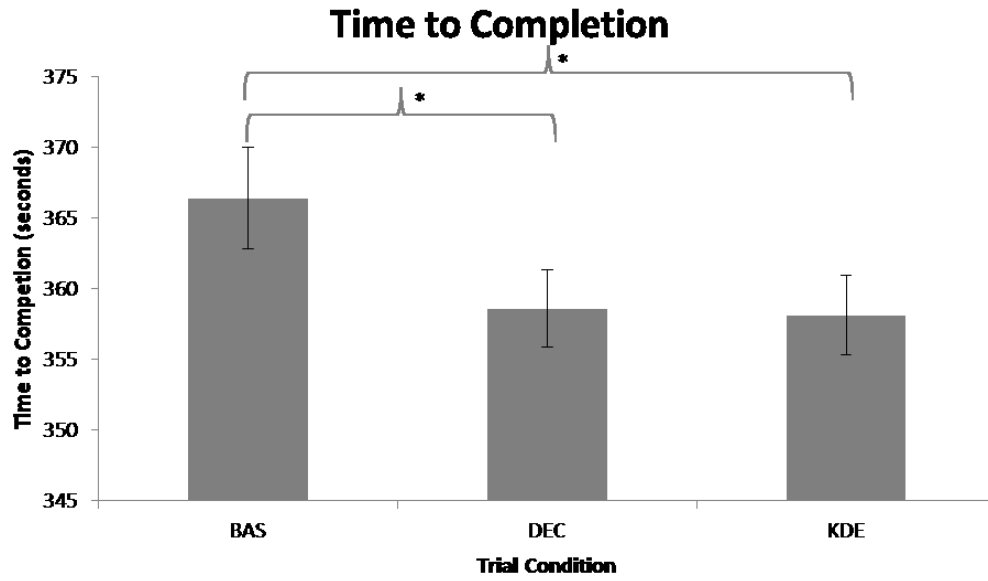


Figure 9. Mean time to completion in all trials for subjects who improved from the BAS to DEC trials. Significant differences from BAS are denoted by an asterisk (* $p < 0.05$).

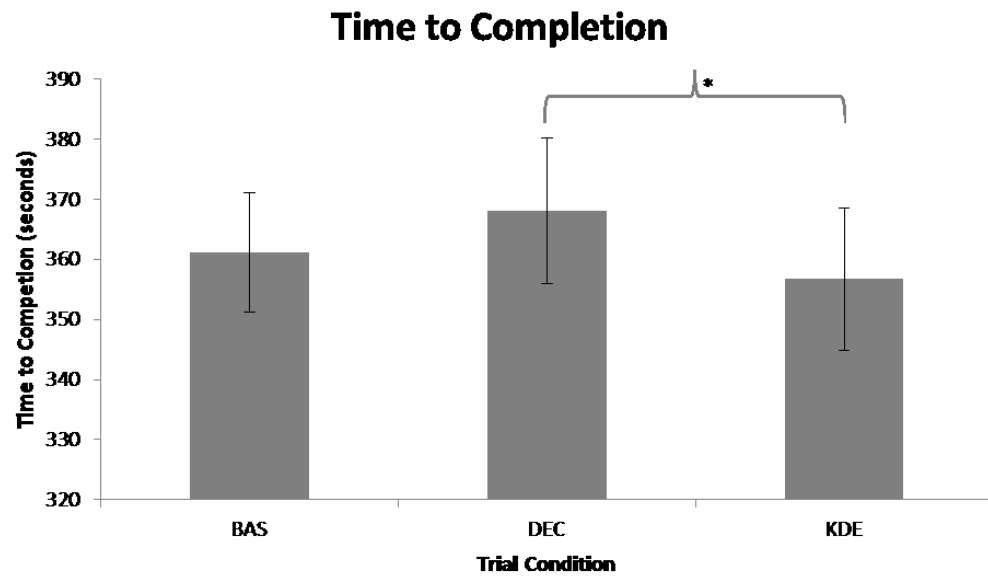


Figure 10. Mean time to completion in all trials for subjects who did not improve from the BAS to DEC trials. Significant differences from DEC are denoted by an asterisk (* $p < 0.05$).

Pacing

No differences were observed in the pacing strategy adopted between trials by subjects who improved in the DEC trial ($F = 1.53, p = 0.238$). Mean power output for this group for each 0.5-km segment was not significantly different between trials (all $p > 0.05$). Similarly, no significant differences were observed in the pacing strategy adopted by subjects who did not improve in the DEC trial ($F = 8.91, p = 0.058$). No differences in were observed in either group for RPE between trials ($F = 1.13, p = 0.346$ for subjects who improved in the DEC trial and $F = 2.63, p = 0.152$ for subjects who did not improve in the DEC trial). Figure 11 displays the mean power output at each 0.5-km segment for each trial condition for all subjects. Figure 12 displays the mean RPE for each kilometer of distance covered for all subjects in all trial conditions.

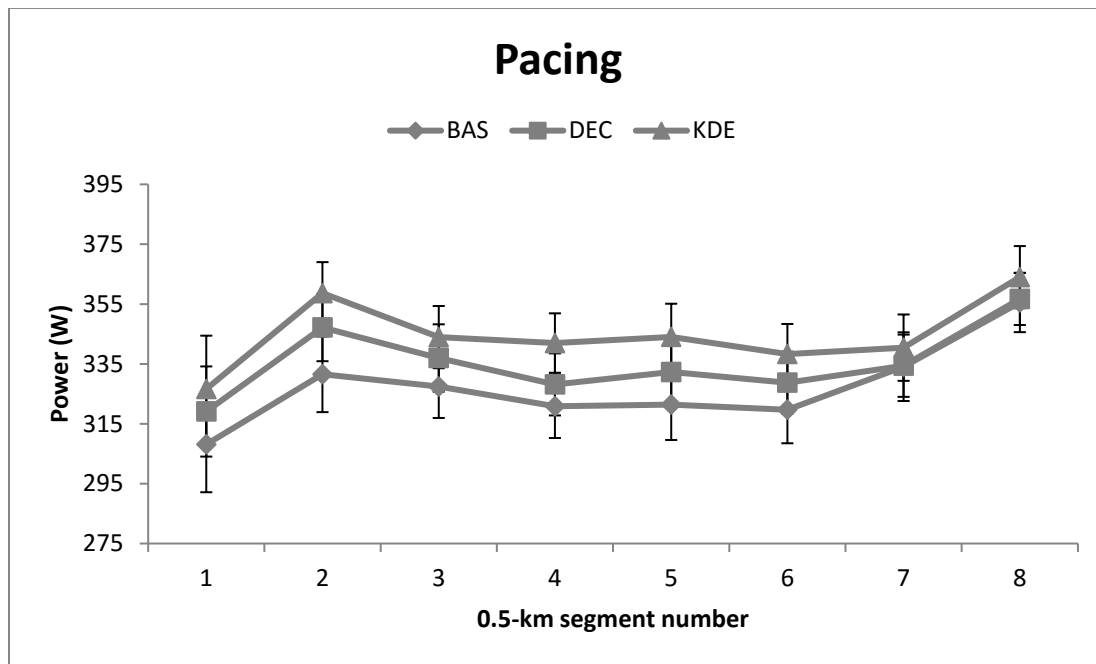


Figure 11. Mean power output for each 0.5-km segment of each trial condition for all subjects. No significant differences were observed.

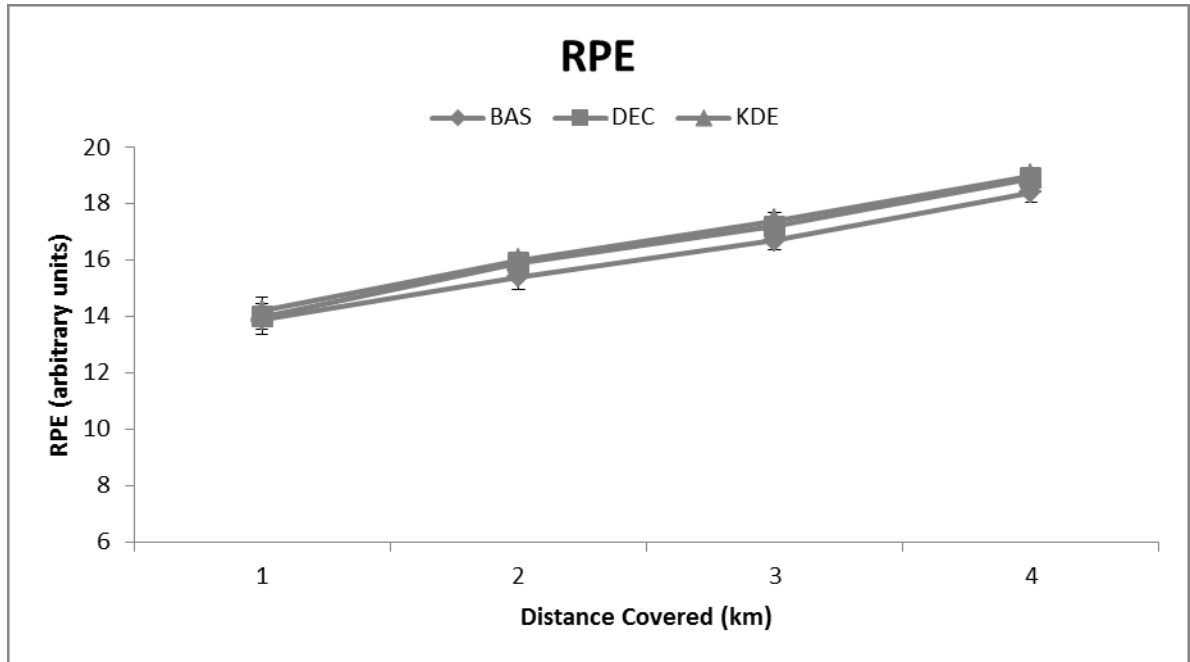


Figure 12. Mean RPE for each kilometer of distance covered for all subjects in all trial conditions. No significant differences were observed.

Self-Motivation

Mean score on the SMI for all subjects was 164.4 ± 14.9 . Mean SMI score for subjects who improved in the DEC trial compared to BAS trial was 165.8 ± 13.3 . Mean SMI score for subjects who did not improve in the DEC trial compared to the BAS trial was 161.3 ± 20.1 . No difference was observed between the two groups for SMI score ($t = 0.417, p = 0.698$). Figure 13 displays a box plot of SMI scores, displayed by min/max, 1st & 3rd quartiles, and median.

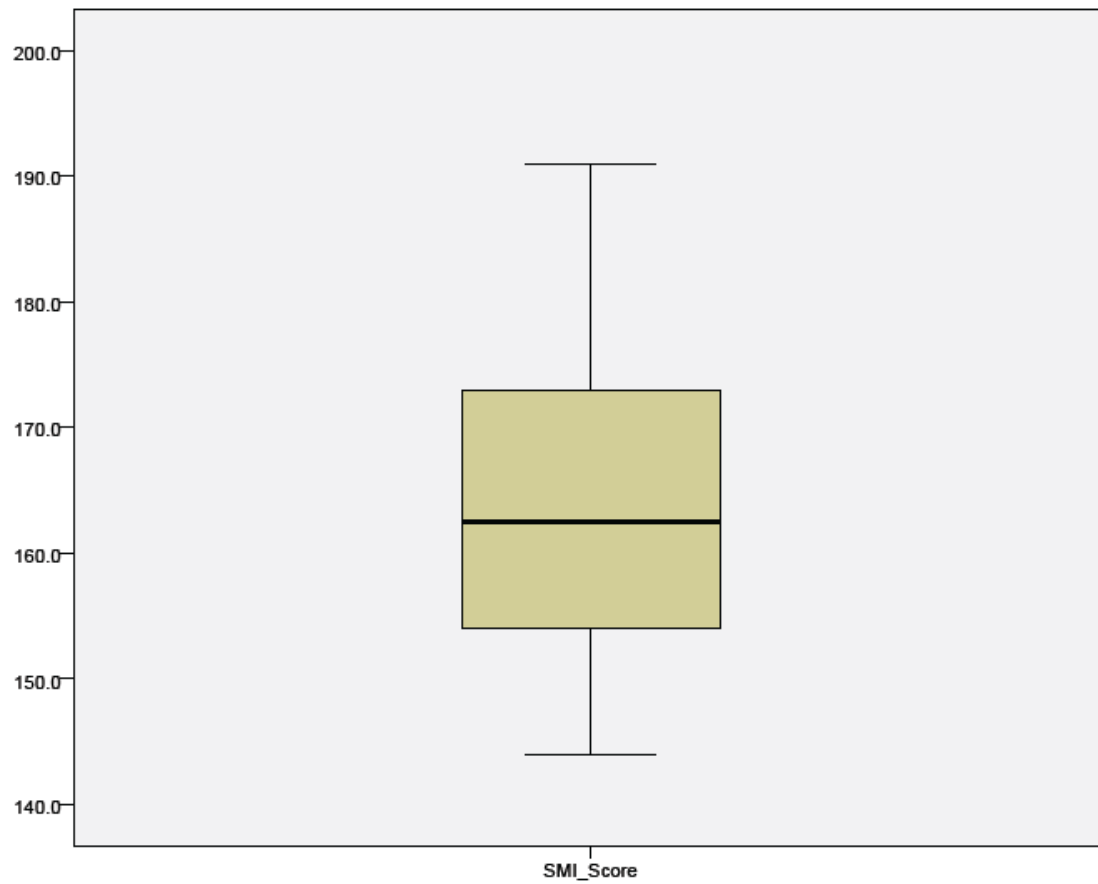


Figure 13. Box plot of SMI scores, displayed by min/max, 1st & 3rd quartiles, and median.

CHAPTER FIVE

DISCUSSION

The aim of this study was to investigate whether subjects who are deceived into improving their performance are able to match that improvement in performance after they are informed of the prior deception. The results of this study indicate that performance remains improved when subjects have knowledge of prior deception. The pacing strategy that is adopted by cyclists does not appear to differ between any of the three experimental conditions (BAS, DEC, KDE trials).

Time Trials

Similar to the data of Stone et al. (Stone, Thomas et al. 2012), the majority of subjects in the present study were able to improve in the DEC trial as compared to the BAS trial. However, the novel finding of the present study was that subjects who improved in the DEC trial compared to the BAS trial performed both the DEC and KDE trials approximately 2.1-2.2% more quickly than the BAS trial, but no differences were observed in time to completion or mean power output between the DEC and KDE trials. There were a number of subjects however, who did not improve in the DEC trial as compared to the BAS trial, but did improve from the DEC trial to the KDE trial. This is in contrast to the findings of Stone et al. (Stone, Thomas et al. 2012), who demonstrated using a randomized and counterbalanced design that cyclists were able to improve their performance when receiving deceptively augmented feedback.

Although Stone et al. (Stone, Thomas et al. 2012) observed an order effect in their study, pilot data ($n = 7$) collected prior to the present study showed no significant order effect between five simulated 4-km time trials for time to completion and mean power output ($F = 0.14$, $p = 0.966$, and $F = 0.13$, $p = 0.969$, respectively). The design of the present study used a fixed order

of trials, which is contrary to the standard practice of randomizing and counterbalancing treatment order in order to negate the effect of order (Lucas 1992). This fixed order was necessary however, since the DEC trial was based on performance in the BAS trial and the KDE trial was dependent on subjects being deceived previously (in the DEC trial). Based on pilot data using the same population as the present study and previous work describing the consistency of time trial performance and pacing across three simulated 4-km cycling time trials (Stone, Thomas et al. 2011), it was concluded that trial order did not affect performance or pacing strategy. Additionally, the assumption that a single familiarization trial improves reliability in subsequent trials has been supported previously (Noreen, Yamamoto et al. 2010, Stone, Thomas et al. 2011).

The lack of change in RPE between trials that was observed in the present study is also contrary to the findings of Stone et al. (Stone, Thomas et al. 2012). This is particularly interesting given the fact that subjects were able to improve in the DEC and KDE trials compared to the BAS trial. It should be noted that while RPE was recorded at each kilometer of the distance completed for each trial, subjects were not asked to provide a whole-trial RPE, which is the method that was used in the Stone et al. study (Stone, Thomas et al. 2012). However, the present study observed no differences between all trials in RPE at each kilometer or mean RPE for each trial in either group, which may be due to the difference in when the measurement tool (Borg's RPE scale, (Borg 1982)) was administered. It is interesting that even in the trials in which subjects performed significantly better, their RPE was not elevated. This suggests that subjects exercised at the same level of exertion, but that their performance level for that same level of exertion was improved, which may be due to the presence of the on-screen avatar providing competition.

Competition has been shown to elicit performance gains in simulated 2-km cycling time trials (Corbett, Barwood et al. 2012), which may account for the improved performance without an observed change in RPE. The influence of competition alone versus deception on performance improvements in simulated 4-km cycling time trials has been previously investigated (Stone, Thomas et al. 2012, Stone, Thomas et al. 2012), illustrating that while competition alone (in the first Stone et al. study (Stone, Thomas et al. 2012), the “ACC” condition) can elicit performance improvements over baseline, deception can elicit further gains that are significantly better than the gains elicited by competition alone. The second study by Stone et al. (Stone, Thomas et al. 2012) demonstrated no change in performance when subjects competed against an avatar set to 102% and 105% of their baseline performance, which is in contrast to their previous finding that subjects improved when they competed against an avatar set at 100% of their baseline performance. A study by Corbett et al. (Corbett, Barwood et al. 2012) did not report RPE, so it is unclear whether subjects consciously exerted more effort when faced with head-to-head competition. The second study by Stone et al. (Stone, Thomas et al. 2012) reported no change in RPE between trials; however performance did not change either, so the implications for the present study where subjects improved without a change in RPE are limited.

The lack of change in RPE is consistent with the finding that power output in each 0.5-km segment of the time trial was not significantly different between trials. Although gross efficiency and aerobic/anaerobic contributions to mechanical power output were not measured, the mechanical power output for each subject was recorded throughout the trial and analyzed for pacing by averaging power output over each 0.5-km of each time trial. The consistency of average power output for each segment of each trial across each trial suggests that subjects

adopted a consistent pacing strategy across all trials, even when their performance was significantly different. Since the pacing was consistent, it is reasonable that RPE throughout the trial would remain consistent as well, which is what was observed in the present study.

The consistency of pacing observed in the present study is consistent with the findings of Stone et al. (Stone, Thomas et al. 2011), who showed that performance and pacing in repeated 4-km time trials is consistent in normal conditions. The present study supports this finding with the addition that neither deceptive feedback, nor knowledge of prior deceptive feedback, appears to alter pacing. One shortcoming of the present study was the lack of quantification of aerobic and anaerobic contributions to mechanical power output, which would provide further insight into the distribution of bioenergetic resources throughout the exercise bout. However, the observation that mechanical power output distribution was not different between trials in subjects who did not improve in the DEC trial provides cursory evidence that pacing is consistent. One of the drawbacks of the estimation of aerobic and anaerobic contributions to mechanical power output is that it assumes that gross efficiency is the same during low-intensity, steady-state cycling as it is during high-intensity, self-paced cycling. This assumption may not be valid (Ettema and Lorås 2009), as it has been demonstrated that gross efficiency decreased throughout the course of a 4-min high-intensity time trial (Uitslag, Galiart et al. 2010). Despite this, previous research has demonstrated that the calculation of anaerobic energy expenditure can still be made without meaningful error (Gastin, Costill et al. 1995). It is yet unclear how valid this measurement is, and thus the cursory examination of total mechanical power output is adequate for the present study for the characterization of pacing.

The ability to improve performance through surreptitiously augmented feedback is already established (Stone, Thomas et al. 2012), and it has been shown that the improvement is

indeed due to the deception itself (Stone, Thomas et al. 2012), but the present study is the first to demonstrate that the performance improvements elicited in this fashion persist once cyclists are informed that they have been previously deceived. The findings of the present study are consistent with those of Noreen et al. (Noreen, Yamamoto et al. 2010), who demonstrated that simulated time trial performance indoors on a Velotron cycle ergometer is repeatable when given constant feedback. Their findings support the consistency between the DEC and KDE trials, as they demonstrated no significant difference between their 2nd and 3rd time trial. As Stone et al. (Stone, Thomas et al. 2012) note, the margin between winning a gold and silver medal in events ranging from 1000- to 4000-m is only ~1.0% in elite competitions (Foster, Schrager et al. 1994, Stone, Thomas et al. 2012), so the persistence of the performance improvement elicited by deception may have positive implications on competitive performance in cyclists.

One of the mechanisms by which Stone et al. (Stone, Thomas et al. 2012) suggested their subjects were able to improve in their deception study was the believed assurance of having completed the same task previously (since the subjects believed that the DEC trial was their own best performance). This mechanism may apply to the DEC and KDE trials as well, since subjects were informed that they successfully improved their performance and that the on-screen avatar in the KDE trial would reflect their “new max,” i.e., 102% of their baseline performance. The deception debriefing script was written to downplay the deceptive nature of the trials and focus on the subjects’ improvement and the emergence of their “new max” in the DEC trial. This assurance that they had previously successfully completed the time trial at the target intensity may have allowed the subjects to exert greater performance and is consistent with the suggestions of Stone et al. (Stone, Thomas et al. 2012).

The data from the present study demonstrates that cyclists carry a reserve capacity even during maximal self-paced exercise (i.e., a time trial). The use of deception to access this reserve is well-characterized (Ness and Patton 1979, Morton 2009, Corbett, Barwood et al. 2012, Stone, Thomas et al. 2012), which effectively resets the maximal capacity to allow for improved performance (i.e., higher mechanical power output). The present study is the first to demonstrate that this resetting of the maximal capacity is retained beyond deceptive trials. If in fact exercise intensity is regulated centrally by a central governor, then the use of deception may be an effective tool in increasing the maximal limit that is allowed by the central governor. This is manifested in cyclists by performance improvements that are retained after prior deception is revealed.

Although the present study demonstrated that performance improvements elicited by deception are retained, it is unclear why subjects who did not improve in the DEC trial were able to improve in the KDE trial. One possible explanation is that these cyclists operate at a higher “relative maximum” than their peers who were able to improve. If cyclists do indeed operate at a relative maximum, perhaps these cyclists have a narrower gap between their absolute and relative maximums compared with cyclists who are able to improve with deception. This is further supported by the fact that these riders improved from the DEC to the KDE trials, but no differences were observed between the BAS and KDE trials. However, the small sample size ($n = 4$) may have been insufficient to observe changes from the BAS to the KDE trials, so interpretation of these data should be done with caution. Further investigation on this topic is warranted before any conclusions can be drawn. The direct effect of head-to-head competition may be disregarded, as the on-screen avatar in the DEC and KDE trials was identical for each subject. The difference between these conditions was the subjects’ knowledge of whether the

feedback given by the avatar was deceptive or not. The knowledge that the on-screen avatar in the KDE trial was set to 102% of their baseline may have served as a motivating factor for these subjects, allowing them to improve their performance. This is in contrast to the findings of Stone et al. (Stone, Thomas et al. 2012) in a follow-up study to their novel deception study (Stone, Thomas et al. 2012) in which they demonstrated that subjects who are informed that their competitors are at 102% or 105% of their baseline performance do not improve. However, the present study included a deception trial prior to the trial in which subjects were informed that they were in fact competing against an avatar set 2% above their baseline performance. Subjects who did not improve in the DEC trial against the faster avatar may have had additional motivation to beat the avatar when they knew they had been beaten by it in the past. The previous experience against the avatar may have influenced their performance as well. Further investigation in this area is warranted to elucidate the mechanisms by which subjects who do not improve when deceived are able to improve their performance when they are informed of the prior deception.

The findings of the present study also support the findings of Mauger et al. (Mauger, Jones et al. 2010), who concluded that learned pacing schemas are robust and not negatively affected by subsequent pacing variation. They also postulated that because their subjects were highly trained cyclists, they may have been able to use their prior experiences to create an optimal pacing strategy in a feed-forward manner. While the subjects in the present study were not elite cyclists, they were experienced competitive cyclists who all had over one year of competitive cycling experience and who were actively engaged in a cycling training regimen. These two factors may have contributed to the robustness of their pacing schemas, as described previously by Mauger et al. (Mauger, Jones et al. 2009, Mauger, Jones et al. 2010). This is

further supported by the findings of Williams et al. (Williams, Bailey et al. 2012), who demonstrated that novice cyclists do not optimize their pacing strategy based on distance feedback like experienced cyclists do.

Self-Motivation

No difference was observed between subjects who did and did not improve from the BAS to the DEC trials. Mean scores on the SMI for both groups was consistent with scores observed in highly motivated athletes (Raglin, Morgan et al. 1990). A previous study on female collegiate rowers found that mean SMI scores for rowers who did not adhere to training for an entire season was significantly lower than that observed for rowers who adhered to training for the entire season (Raglin, Morgan et al. 1990). Mean scores for the groups from the study by Raglin et al. (Raglin, Morgan et al. 1990) is shown in Figure 14.

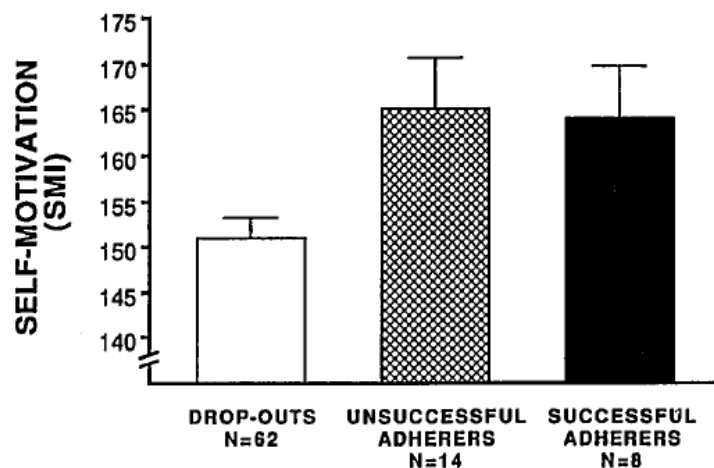


Figure 2—Self-motivation (SMI) scores (mean \pm SE) for dropouts, unsuccessful adherers, and successful adherers.

Figure 14. Mean SMI scores for collegiate female rowers, separated by group – dropouts who did not adhere to training for an entire season, unsuccessful adherers who adhered to training for an entire season but were not chosen to compete in the final competition, and successful adherers who adhered to training for an entire season and were chosen to compete in the final competition. (Raglin, Morgan et al. 1990)

Mean scores for all subjects in the present study are consistent with that of both adherer groups in the Raglin et al. study ((Raglin, Morgan et al. 1990). Since there was no difference observed in SMI score between groups, it is unlikely that subjects who did not improve from the BAS to the DEC trials did not improve because they were less motivated than those who did improve from the BAS to the DEC trials. One shortcoming that should be noted is the small sample size of subjects who did not improve from the BAS to the DEC trials. More subjects are needed in this group to be able to draw meaningful conclusions about the correlation between motivation and improvement from BAS to DEC trials.

Future Directions

The present study has demonstrated that after subjects are deceived into improving their performance via surreptitiously augmented feedback in the form of an on-screen avatar set to 102% of their baseline power output, they are able to retain that performance improvement after they are informed of the deception. Further research is needed to elucidate whether there are similar changes in their aerobic and anaerobic contributions to total power output once they are informed of the deception as are observed from baseline to deception (Stone, Thomas et al. 2012). Furthermore, future research should aim to elucidate whether or not subjects are able to retain their improved performance in a maximal self-paced time trial with *no* on-screen avatar following deception and debriefing. Additional research is also needed to investigate why some cyclists do not improve when deceived. To investigate more about fatigue, future research may focus on quantifying central and peripheral fatigue following each time trial and elucidating whether there are significant changes in central and peripheral fatigue following baseline, deception, and knowledge of deception time trials.

Conclusions

In summary, this is the first study to demonstrate that performance gains elicited in cyclists using deception are retained after the deception is revealed. The improvement in performance is achieved without changes in pacing strategy or RPE throughout the time trial. These findings support the notion that cyclists normally operate with a reserve capacity during maximal time-trial performances. These findings also indicate that deception may be used to access this reserve and that through deception the maximal capacity of cyclists is improved, suggesting a re-setting of the metabolic reserve capacity. This has implications on methods of enhancing performance in trained cyclists and the regulation of fatigue and pacing during maximal time trial exercise. These findings further demonstrate the complexity of fatigue development and regulation during maximal self-paced exercise.

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APPENDIX A
INFORMED CONSENT STATEMENT

INDIANA UNIVERSITY INFORMED CONSENT STATEMENT FOR

Consistency and pacing strategy during repeated 4-km cycling time trials

You are invited to participate in a research study of consistency and pacing strategy during repeated 4-kilometer cycling time trials. You were selected as a possible subject because you are a competitive cyclist. We ask that you read this form and ask any questions you may have before agreeing to be in the study.

Disclaimer: It is possible that you will not qualify for the study following the submission of exercise logs and completion of the $\text{VO}_{2\text{max}}$ test.

The study is being conducted by Dr. Timothy D. Mickleborough and Ren-Jay Shei with the Indiana University/Department of Kinesiology.

STUDY PURPOSE

The purpose of this study is to investigate whether 4-kilometer cycling time trial performance during repeated trials is consistent and also to assess the pacing strategy during repeated 4-kilometer cycling time trials.

NUMBER OF PEOPLE TAKING PART IN THE STUDY:

If you agree to participate, you will be one of twenty (20) subjects who will be participating in this research.

PROCEDURES FOR THE STUDY:

If you agree to be in the study, you will do the following things:

You will visit the Human Performance Laboratory a total of five (5) times. Each visit will be done at a previously agreed-upon time. The first visitation will last approximately 90 minutes and the remaining four will last approximately 75 minutes. You should refrain from exhaustive exercise for 24 hours prior to each visit. You should also avoid consuming caffeine and alcohol for 24 hours prior to each visit. Upon arriving at the laboratory you will be asked to complete a consent form, questionnaire (PAR-Q), and training log. You will then assume a rested position, seated on a cycle ergometer (stationary bike). During that time you will be asked to breathe into a face-mask that allows for the collection of exhaled gas. The mask will be fitted to your face and adjusted for comfort. The face-masks are cleansed in a detergent solution and submerged in an antibacterial solution following each use. After the resting period is complete, you will be asked to begin cycling for the maximal aerobic capacity test. Before and after each exercise bout, blood pressure will be measured in order to monitor for potential hypotension.

Maximal Aerobic Capacity Test ($\text{VO}_{2\text{max}}$ test)

During the first visit you will be asked to perform a graded exercise test to measure maximal oxygen consumption. This test will be completed on a cycle ergometer. Prior to the cycling exercise bout, the seat and handlebars of the ergometer will be adjusted in order to accommodate your preferred cycling posture. You will be allowed to bring in your own pedals, shoes, and saddle if you desire. Otherwise, the stock saddle and pedals will be used on the cycle ergometer. Resting measurements will be collected for

5 minutes following brief warm-up. You will begin cycling at a cadence of your choice (for example, 90 rpm) at a workload of 100 Watts (W). Every 1 min, 25 W of workload will be added until you can no longer maintain the required power output. Following the final stage, you will have the opportunity to cool down on the cycle ergometer.

During this exercise test you will be breathing through rubber face-mask fixed to a one-way, non-rebreathing valve. Room air will flow through the tube on the inspired side and into your lungs. Expired air will flow through the other tube and into a fiberglass chamber. From the chamber, a pump constantly pulls air into analyzers to determine the composition of the expired gas.

If you qualify for the study (as determined by your answers on the questionnaire and training log as well as the results of the maximal aerobic capacity test), you will be admitted into the study and asked to return to the laboratory for four (4) more tests.

4-km Time Trial

This test consists of a self-paced, maximal effort for a computer-simulated 4-kilometer cycling time trial on an electromagnetically braked cycle ergometer interfaced with computer software. You will complete four of these time trials on separate occasions. In this study, standard road gearing will be available to you and the course will be a flat, 4-kilometer course. The software also allows for a computer-generated avatar to be displayed, simulating head-to-head competition. This feature will be utilized in the third and fourth time trial. Each of these sessions will require approximately 75 minutes total, with approximately 5-8 minutes of exercise.

Prior to the start of each trial and immediately upon completion of each trial, capillary blood samples will be taken by pricking your finger.

RISKS OF TAKING PART IN THE STUDY:

In general, risk associated with participation in this study is minimal. Risks for individual tests and procedures are listed below:

Submaximal and maximal exercise presents little to no risk in healthy asymptomatic individuals under the age of 40 years, as described by the American College of Sports Medicine. However the possible discomforts involved with exercise testing can include episodes of temporary lightheadedness, chest discomfort, leg cramps, significant leg fatigue, nausea, occasional irregular heart-beats, and abnormal blood pressure responses. Non-maximal exercise poses little risk to the subject.

During cycle ergometry there is a risk of blood pooling in the lower extremities. This can lead to hypotension, light-headedness, and dizziness following exercise. In addition, there is a possibility of hypotension following maximal exercise testing.

Maximal Aerobic Capacity Exercise Tests: A maximal exercise test of healthy individuals, as described by the American College of Sports Medicine, or a submaximal exercise bout, presents little risk to the subject and does not require medical clearance for subjects under 40. Potential risks and/or discomforts can include episodes of transient light-headedness, chest discomfort, leg cramps, occasional irregular heartbeats, and abnormal blood pressure responses. The risk of mortality is approximately 1 in 10,000. During the test you will be closely monitored for any abnormal changes in cardiovascular function. You are free to indicate any discomfort and discontinue participation at any time.

There is a risk that during the exercise intervention, the subject could experience any of the usual symptoms associated with prolonged exercise (dehydration, fatigue, muscle cramping or strain, and the risks listed above for submaximal exercise bouts).

There is a slight risk you will experience light bleeding during the blood collection, however the volume of blood that will be collected during each trial is minimal.

There is a potential risk to the subject of loss of confidentiality.

BENEFITS OF TAKING PART IN THE STUDY:

Individual subjects will receive information about their physical fitness. As highly-trained individuals, participants will benefit from the increase in understanding of their aerobic capacity and how their performance changes throughout the series of 4-km cycling time trials.

ALTERNATIVES TO TAKING PART IN THE STUDY:

An alternative to participating in the study is to choose not to participate.

CONFIDENTIALITY

Efforts will be made to keep your personal information confidential. Data will be stored on password protected computers in locked rooms with limited public access. We cannot guarantee absolute confidentiality. Your personal information may be disclosed if required by law. Your identity will be held in confidence in reports in which the study may be published and databases in which results may be stored.

Organizations that may inspect and/or copy your research records for quality assurance and data analysis include groups such as the study investigator and his/her research associates, the IU Institutional Review Board or its designees, and (as allowed by law) state or federal agencies, specifically the Office for Human Research Protections (OHRP), who may need to access the collected medical and/or research data.

PAYMENT

You will not receive payment for taking part in this study.

CONTACTS FOR QUESTIONS OR PROBLEMS

For questions about the study or a research-related injury, contact the researcher Timothy D. Mickleborough, Ph.D. by phone at 812-855-0753 or by e-mail at tmickleb@indiana.edu. If you cannot reach the researcher during regular business hours (i.e. 8:00AM-5:00PM), please call the IU Human Subjects Office at (812) 856-4242 or (800) 696-2949.

For questions about your rights as a research participant or to discuss problems, complaints or concerns about a research study, or to obtain information, or offer input, contact the IU Human Subjects Office at (812) 856-4242 or (800) 696-2949.

VOLUNTARY NATURE OF STUDY

Taking part in this study is voluntary. You may choose not to take part or may leave the study at any time. Leaving the study will not result in any penalty or loss of benefits to which you are entitled. Your decision whether or not to participate in this study will not affect your current or future relations with the investigators or Indiana University.

SUBJECT'S CONSENT

In consideration of all of the above, I give my consent to participate in this research study.

I will be given a copy of this informed consent document to keep for my records. I agree to take part in this study.

Subject's Printed Name: _____

Subject's Signature: _____ **Date:** _____
(must be

dated by the subject)

Printed Name of Person Obtaining Consent: _____

Signature of Person Obtaining Consent: _____ **Date:** _____

IRB Approval Date: Mar 7, 2013
Expiration Date: Oct 10, 2013

APPENDIX B

MEDICAL QUESTIONNAIRE

Modified Physical Activity Readiness Questionnaire (PAR-Q)

Name			Date
DOB	Age	Home Phone	Work Phone

Regular exercise is associated with many health benefits, yet any change of activity may increase the risk of injury. Please read each question carefully and answer every question honestly:

Yes	No	1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
Yes	No	2. Do you feel pain in your chest when you do physical activity?
Yes	No	3. In the past month, have you had chest pain when you were not doing physical activity?
Yes	No	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
Yes	No	5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?
Yes	No	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
Yes	No	7. Do you know of any other reason you should not participate in physical activity?
Yes	No	8. Has your doctor ever told you that you have diabetes?
Yes	No	9. Has your doctor ever told you that you have high blood pressure?
Yes	No	10. Has your doctor ever told you that you have high cholesterol?
Yes	No	11. Has your doctor ever told you that you have high blood sugar?
Yes	No	12. Do you smoke?
Yes	No	13. Are you currently inactive?
Yes	No	14. Do you have a father, brother or son with heart disease before the age of 55 years old or a mother, sister or daughter with heart disease before the age of 65 years old?
15. Measure height and weight to determine BMI: Height: _____		

Weight: _____

Participant Signature	Date
------------------------------	-------------

Note to ParQ Reader:

A “yes” to any Question 1-8 will eliminate the individual from participation.

A “yes” to 2 or more of Questions 9-14 indicates > low risk.

#15: If over 30 kg/m², the individual may have the risk factor of obesity.

APPENDIX C

MOTIVATION AND TRAINING QUESTIONNAIRES

SELF-REPORT QUESTIONNAIRE

NAME _____

DATE _____

Directions: Read each of the following statements and write by each item the letter of the alternative which best describes how characteristic the statement is when applied to you. Please be sure to answer every item and try to be as honest and accurate as possible in your responses. The alternatives are:

- a. extremely uncharacteristic of me
- b. somewhat uncharacteristic of me
- c. neither characteristic nor uncharacteristic of me
- d. somewhat characteristic of me
- e. extremely characteristic of me

- _____ 1. I'm not very good at committing myself to do things.
- _____ 2. Whenever I get bored with projects I start, I drop them to do something else.
- _____ 3. I can persevere at stressful tasks, even when they are physically tiring or painful.
- _____ 4. If something gets to be too much of an effort to do, I'm likely to just forget it.
- _____ 5. I'm really concerned about developing and maintaining self-discipline.
- _____ 6. I'm good at keeping promises, especially the ones I make to myself.
- _____ 7. I don't work any harder than I have to.
- _____ 8. I seldom work to my full capacity.
- _____ 9. I'm just not the goal setting type.
- _____ 10. When I take on a difficult job, I make the point of sticking with it until it's completed
- _____ 11. I'm willing to work for things I want as long as it's not a big hassle for me
- _____ 12. I have a lot of self-motivation
- _____ 13. I'm good at making decisions and standing by them
- _____ 14. I generally take the path of least resistance.

- _____ 15. I get discouraged easily.
- _____ 16. If I tell somebody I'll do something, you can depend on it being done.
- _____ 17. I don't like to overextend myself.
- _____ 18. I'm basically lazy.
- _____ 19. I have a very hard-driving, aggressive personality.
- _____ 20. I work harder than most of my friends.
- _____ 21. I can persist in spite of pain or discomfort
- _____ 22. I like to set goals and work toward them.
- _____ 23. Sometimes I push myself harder than I should.
- _____ 24. I tend to be overly apathetic.
- _____ 25. I seldom if ever let myself down.
- _____ 26. I'm not very reliable.
- _____ 27. I like to take on jobs that challenge me.
- _____ 28. I change my mind about things quite easily.
- _____ 29. I have a lot of will power.
- _____ 30. I'm not likely to put myself out if I don't have to.
- _____ 31. Things just don't matter much to me.
- _____ 32. I avoid stressful situations.
- _____ 33. I often work to the point of exhaustion.
- _____ 34. I don't impose much structure on my activities.
- _____ 35. I never force myself to do things I don't feel like doing.
- _____ 36. It takes a lot to get me going.
- _____ 37. Whenever I reach a goal, I set a higher one.

- _____ 38. I can persist in spite of failure.
- _____ 39. I have a strong desire to achieve.
- _____ 40. I don't have much self-discipline.

Question Number	Score format (a-e)
1.	5 to 1
2.	5 to 1
3.	1 to 5
4.	5 to 1
5.	1 to 5
6.	1 to 5
7.	5 to 1
8.	5 to 1
9.	5 to 1
10.	1 to 5
11.	5 to 1
12.	1 to 5
13.	1 to 5
14.	5 to 1
15.	5 to 1
16.	1 to 5
17.	5 to 1
18.	5 to 1
19.	1 to 5
20.	1 to 5
21.	1 to 5
22.	1 to 5
23.	1 to 5
24.	5 to 1

25.	1 to 5
26.	5 to 1
27.	1 to 5
28.	5 to 1
29.	1 to 5
30.	5 to 1
31.	5 to 1
32.	5 to 1
33.	1 to 5
34.	5 to 1
35.	5 to 1
36.	5 to 1
37.	1 to 5
38.	1 to 5
39.	1 to 5
40.	5 to 1

Indiana University – Human Performance Laboratories - Training Log

- 1) Fill in date for each day
- 2) Indicate the total volume (in miles and/or hours : minutes) of your AM and PM workouts
- 3) Rate the intensity of each workout on a scale of 1 to 5, where
1 = gentle, 2 = moderately gentle, 3 = moderate, 4 = moderately hard, 5 = hard
- 4) Indicate the specifics of the workout session

Date		Volume	Intensity	Workout specifics
Sun	AM			
	PM			
Mon	AM			
	PM			
Tue	AM			
	PM			

Wed	AM			
	PM			
Thur	AM			
	PM			
Fri	AM			
	PM			
Sat	AM			
	PM			

Form Date: 08/15/2012

Indiana University - Human Performance Laboratories - Questionnaire

- 1) How long have you been cycling?
 - a. How long have you been training for competitive cycling?
- 2) Have you competed in a cycling race in the past 12 months?
- 3) Briefly describe your competitive cycling experience.
- 4) Are you currently engaged in competitive cycling training?
 - a. If so, please complete the attached training log

APPENDIX D
DATA COLLECTION SHEETS

RJ Thesis Flight Plan

Pre Subject:

Environmental Conditions: Date: _____

Turn on: ☐ O₂ and CO₂ Analyzers and Flow Control (30min prior) Subject: _____

☐ Velotron _____ Temperature: _____

☐ Computers: DasyLab - Save to flash drive! Humidity: _____

☐ Velotron – Max: CS Max test -OR- 3D Software Pbar: _____

☐ DasyLab VO₂ (metabolics) –OR- TTmetabolics T.Corrected factor: _____

☐ Turn on Pneumotach (inspired) & change Dririte Height: _____

Calibrate: ☐ O₂ and CO₂ Analyzers Weight: _____

Equipment: ☐ Lancet –4 capillary tubes – Gauze – sterile alcohol swabs Post cal O₂: _____

☐ Protective eyewear Post cal CO₂: _____

☐ Lab coat Final Power: _____

☐ Gloves SEAT HEIGHT: _____

☐ Card for capillary tubes; caps for capillary tubes Trial Condition: Max

FAM BAS DEC KDE

☐ RPE chart

☐ Connect hoses: →pneumotach→mouthpiece→ mixing chamber (make sure sample line is in mixing chamber)

☐ Construct mouthpiece/nose clips

☐ Blood pressure cuffs

☐ Heart Rate Monitor

Preparing Subject:

☐ Adjust bike settings

☐ Take blood pressure pre-exercise

☐ Put E.C. in DasyLab VO₂

☐ Decide on RPMs and instruct subject (max only)

☐ Instruct subject to put on HRM

☐ Collect initial capillary blood sample (two tubes; TT only)

☐ Get resting VO₂ Data (3-5 mins)

	Pre-Exercise	Post-Exercise
Blood Pressure		

During Trials:

☐ RPE every 1km

☐ No verbal encouragement

☐ Collect post-exercise capillary blood sample (2 tubes, ASAP after TT)

☐ Save CS file (F5 on controller to “prompt save, reset, cancel”)

☐ Take blood pressure post-exercise

RPE	KM 1	
	KM 2	
	KM 3	
	KM 4	

Clean up:

- ☐ Clean mouth piece
- ☐ Disconnect hoses
- ☐ Save data to flash drive! (export metabolic data to Excel (all trials); save Velotron CS ergometer test (max only); save Velotron report and rider performance and export performance to Excel)

*How to Name Files:

VO₂: Initials(max)

Test type: Familiarization trial = FAM; or other conditions: BAS, DEC,

KDE,

Ex: JWmax = JW $\dot{V}O_2$ max

Ex: RSBAS= RS Baseline trial

Trial Condition	FAM	BAS	DEC	KDE
Time to Completion				
Mean Power				
Mean Speed				

APPENDIX E
POWER ANALYSES

Stone, M.R., K. Thomas, M. Wilkinson, A.M. Jones, A. St Clair Gibson, and K.G. Thompson. Effects of deception on exercise performance: implications for determinants of fatigue in humans. *Med. Sci. Sports Exerc.*, Vol. 44, No. 3, pp.534–541.

Time to completion

[1] -- Tuesday, December 04, 2012 -- 12:05:36

F tests – ANOVA: Repeated measures, within factors

Analysis: A priori: Compute required sample size

Input:	Effect size f	=	0.8912720
	α err prob	=	0.05
	Power (1- β err prob)	=	0.95
	Number of groups	=	1
	Number of measurements	=	2
	Corr among rep measures	=	0.5
	Nonsphericity correction ϵ	=	1
Output:	Noncentrality parameter λ	=	22.2422418
	Critical F	=	5.9873776
	Numerator df	=	1.0000000
	Denominator df	=	6.0000000
	Total sample size	=	7
	Actual power	=	0.9727656

Mean power output

[2] -- Tuesday, December 04, 2012 -- 12:10:51

F tests – ANOVA: Repeated measures, within factors

Analysis: A priori: Compute required sample size

Input:	Effect size f	=	0.8164966
	α err prob	=	0.05
	Power (1- β err prob)	=	0.95
	Number of groups	=	1
	Number of measurements	=	2
	Corr among rep measures	=	0.5
	Nonsphericity correction ϵ	=	1
Output:	Noncentrality parameter λ	=	21.3333343
	Critical F	=	5.5914479
	Numerator df	=	1.0000000
	Denominator df	=	7.0000000
	Total sample size	=	8
	Actual power	=	0.9753507

RPE

[3] -- Tuesday, December 04, 2012 -- 12:11:59

F tests – ANOVA: Repeated measures, within factors

Analysis: A priori: Compute required sample size

Input:	Effect size f	=	0.6925448
	α err prob	=	0.05
	Power (1- β err prob)	=	0.95
	Number of groups	=	1
	Number of measurements	=	2
	Corr among rep measures	=	0.5

	Nonsphericity correction ϵ	=	1
Output:	Noncentrality parameter λ	=	17.2662588
	Critical F	=	5.3176551
	Numerator df	=	1.0000000
	Denominator df	=	8.0000000
	Total sample size	=	9
	Actual power	=	0.9516233

Pacing

[4] -- Tuesday, December 04, 2012 -- 12:13:05

F tests – ANOVA: Repeated measures, within factors

Analysis: A priori: Compute required sample size

Input:	Effect size f	=	0.5426771
	α err prob	=	0.05
	Power (1- β err prob)	=	0.95
	Number of groups	=	1
	Number of measurements	=	2
	Corr among rep measures	=	0.5
	Nonsphericity correction ϵ	=	1
Output:	Noncentrality parameter λ	=	16.4919124
	Critical F	=	4.6671927
	Numerator df	=	1.0000000
	Denominator df	=	13.0000000
	Total sample size	=	14
	Actual power	=	0.9629734

APPENDIX F

RAW DATA – MEAN POWER AND TIME TO COMPLETION

Subject	FAM_TTC	FAM_Mean Power	BAS_TTC	BAS_Mean_Power	DEC_TTC	DEC_Mean_Power	KDE_TTC	KDE_Mean_Power
S01	356.61	345.3	356.40	343.8	351.96	356.7	350.52	358.0
S02	350.94	356.6	344.51	376.2	348.21	367.4	340.17	389.3
S03	341.10	393.3	346.19	375.7	350.76	362.5	336.75	402.0
S04	353.19	353.8	354.83	348.6	350.60	358.2	350.63	360.2
S05	388.35	271.4	386.85	273.4	400.39	250.9	388.10	271.7
S06	388.29	271.6	381.14	285.1	375.18	298.1	379.19	303.3
S07	355.73	347.9	369.18	315.2	359.04	341.1	353.00	352.6
S08	364.89	325.1	366.92	315.5	373.11	302.6	361.98	330.0
S09	360.17	334.6	362.39	328.8	354.12	347.2	357.22	341.0
S10	350.30	358.5	351.84	356.0	351.11	359.1	354.48	346.6
S11	366.80	318.6	363.57	326.6	356.73	343.6	361.47	329.2
S12	376.07	315.5	372.66	309.4	360.06	336.3	355.61	348.3
S13	362.66	327.3	387.11	277.5	372.02	306.3	366.38	316.7
S14	364.14	333.1	364.91	326.0	355.29	346.9	352.73	355.8
Mean	362.80	332.33	364.89	325.56	361.33	334.06	357.73	343.19
StDev	13.745	32.603	13.699	32.837	14.353	32.928	13.597	33.000
CV	0.038	0.098	0.038	0.101	0.040	0.099	0.038	0.096

APPENDIX G

RAW DATA – PACING AND RPE

Subject	BAS_Seg_1	BAS_Seg_2	BAS_Seg_3	BAS_Seg_4	BAS_Seg_5	BAS_Seg_6	BAS_Seg_7	BAS_Seg_8
S01	350.5	413.2	367.8	323.8	331.9	323.6	333.2	315.9
S02	329.9	367.9	378.2	376.0	385.7	391.2	395.7	397.0
S03	271.1	366.1	380.6	403.8	395.2	384.2	409.3	428.1
S04	290.8	362.7	366.3	363.6	352.2	356.2	358.0	352.4
S05	258.0	283.9	270.7	271.1	271.3	269.9	266.8	300.9
S06	291.4	292.4	285.2	284.4	273.0	273.1	267.8	316.9
S07	412.9	288.2	277.6	300.7	292.4	290.7	303.8	370.1
S08	301.8	330.9	322.8	310.4	311.5	305.2	312.2	333.6
S09	328.1	318.6	328.9	325.2	340.3	307.9	338.8	346.2
S10	422.2	386.7	353.0	343.0	337.5	337.1	335.9	337.7
S11	339.7	362.0	345.9	304.8	283.4	296.2	338.1	352.5
S12	247.3	338.7	310.4	281.8	282.7	295.8	344.2	402.4
S13	234.3	276.0	276.8	275.8	269.2	272.6	295.0	334.7
S14	235.7	254.2	319.7	327.6	373.4	372.1	378.5	389.1

Subject	DEC_Seg_1	DEC_Seg_2	DEC_Seg_3	DEC_Seg_4	DEC_Seg_5	DEC_Seg_6	DEC_Seg_7	DEC_Seg_8
S01	305.6	378.6	371.4	371.3	369.2	356.4	351.2	361.8
S02	339.4	403.1	397.7	389.6	388.9	350.1	346.9	336.0
S03	328.8	394.9	383.5	351.2	349.4	343.7	356.4	402.7
S04	308.7	370.9	371.3	366.4	370.5	364.3	358.8	365.8
S05	204.7	240.3	243.9	259.1	258.2	262.2	271.9	279.5
S06	335.1	339.6	298.9	276.1	288.4	264.7	276.0	313.3
S07	433.5	331.7	325.8	317.2	319.6	313.3	339.6	354.5
S08	304.1	316.4	307.5	289.2	285.1	285.4	298.1	340.4
S09	293.0	353.4	353.2	354.4	358.7	351.0	362.4	362.7
S10	416.2	396.1	338.6	338.6	326.5	363.4	339.2	360.2
S11	321.4	332.6	372.8	330.2	317.4	349.6	365.2	368.4
S12	265.1	322.7	322.6	330.4	381.1	368.7	356.8	363.9
S13	320.8	333.6	292.4	283.9	297.4	282.5	269.5	382.2
S14	291.2	346.5	338.1	336.7	341.8	346.9	389.6	402.2

Subject	KDE_Seg_1	KDE_Seg_2	KDE_Seg_3	KDE_Seg_4	KDE_Seg_5	KDE_Seg_6	KDE_Seg_7	KDE_Seg_8
S01	307.3	396.4	384.9	372.7	355.1	369.5	355.0	336.4
S02	339.8	406.6	401.2	406.6	417.2	378.1	383.1	394.3
S03	332.9	419.8	421.7	407.1	399.3	406.3	419.7	425.9
S04	312.1	370.8	367.4	368.1	366.3	364.4	358.4	386.7
S05	242.1	282.7	282.6	277.6	269.8	270.9	271.6	283.4
S06	258.4	402.4	304.2	306.7	293.7	295.3	279.5	308.6
S07	508.2	334.2	309.8	332.5	322.8	330.3	339.3	363.4
S08	278.0	337.8	336.1	332.0	332.0	334.5	333.1	369.3
S09	313.7	326.5	337.8	331.1	361.5	348.9	355.7	360.7
S10	416.9	374.2	331.2	338.7	337.3	325.1	317.5	338.6
S11	350.7	365.3	340.7	303.5	306.6	310.3	305.9	358.7
S12	296.5	343.4	348.9	352.5	358.4	326.4	371.3	404.0
S13	311.8	323.9	307.4	317.6	311.6	303.3	304.0	360.7
S14	304.3	337.2	341.5	341.0	384.9	377.4	372.1	405.3

Subject	BAS_RPE_KM1	BAS_RPE_KM2	BAS_RPE_KM3	BAS_RPE_KM4
S01	18	19	18	19
S02	15	16	18	20
S03	12	13	14	16
S04	13	15	17	19
S05	10	13	15	18
S06	13	15	17	19
S07	14	15	16	17
S08	15	16	17	19
S09	12	13	15	16
S10	17	17	18	19
S11	15	16	17	18
S12	14	15	17	18
S13	14	16	17	20
S14	13	16	18	19

Subject	DEC_RPE_KM1	DEC_RPE_KM2	DEC_RPE_KM3	DEC_RPE_KM4
S01	18	18	19	19
S02	15	17	18	20
S03	14	16	16	18
S04	13	15	17	19
S05	12	15	17	19
S06	14	16	17	19
S07	13	15	17	20
S08	15	16	17	18
S09	12	14	15	16
S10	16	17	17	19
S11	12	15	18	20
S12	13	16	18	20
S13	15	16	17	19
S14	14	16	18	19

Subject	KDE_RPE_KM1	KDE_RPE_KM2	KDE_RPE_KM3	KDE_RPE_KM4
S01	19	19	20	20
S02	15	17	19	20
S03	12	15	17	18
S04	12	15	18	19
S05	14	16	18	20
S06	14	16	17	19
S07	13	15	17	19
S08	15	16	17	18
S09	13	15	16	16
S10	14	16	17	19
S11	13	15	16	19
S12	15	17	18	20
S13	16	16	17	20
S14	14	16	17	19

APPENDIX H
RAW DATA – SMI SCORES

Subject	SMI_Score
S01	167
S02	152
S03	144
S04	154
S05	158
S06	156
S07	145
S08	190
S09	168
S10	173
S11	157
S12	191
S13	178
S14	169

Curriculum Vitae

Ren-Jay Shei

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Indiana University

Department of Kinesiology

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EDUCATION

Master of Science in Kinesiology (2013)

Major: Exercise Physiology

Indiana University – Bloomington

Thesis: Deception and exercise performance during repeated 4-km cycling time trials.

Bachelor of Science in Kinesiology (2011)

Major: Exercise Science

Minor: Coaching

Indiana University – Bloomington

Bachelor of Science in Biology (2011)

Minors: Chemistry, Social Science and Medicine

Indiana University – Bloomington

PROFESSIONAL EXPERIENCE

Associate/Adjunct Instructor: Indiana University

K535 Laboratory Instructor: Physiological Basis of Human Performance

X590 Associate Instructor: Introduction to Research in Health, Kinesiology, and Recreation

K409/P409 Laboratory Instructor: Basic Physiology of Exercise

E112 Lecture Instructor: Bicycling

E168 Activity Instructor: Beginning Swimming

E268 Activity Instructor: Intermediate Swimming

K535 Lecturer: Physiological Basis of Human Performance (Invited Lecture: Structure and Function of Exercising Muscle)

N530 Grader: Advanced Human Nutrition

Attendee, Teaching Research Ethics Workshop at Indiana University by the Poynter Center for the Study of Ethics and American Institutions

RESEARCH

My area of research is focused on the relative contributions of central and peripheral factors to fatigue. I am currently investigating the effects of deception on integrative central control during maximal exercise, specifically during 4-kilometer cycling time trials. The central governor model of integrative central control proposes that exercise intensity is regulated by the subconscious brain and that peripheral signals and conscious input are integrated, resulting in a continuous modification of motor drive to working muscles. This regulation maintains homeostasis and thus is a protective mechanism to prevent catastrophic physiological events such as hyperthermia and ischemia from occurring.

PUBLICATIONS

Shei, R-J. and Mickleborough, T.D. Relative contributions of central and peripheral factors in human muscle fatigue during exercise. (submitted – under review *Journal of Sports Medicine*)

Mickleborough, T.D., Vaughn, C., **Shei, R-J.**, Davis, E., and Wilhite, D.P. Marine Lipid fraction PCSO-524™ (Lyprinol®/Omega XL®) of the New Zealand green lipped mussel attenuates hyperpnea-induced bronchoconstriction in asthma. (In Press - *Respiratory Medicine*)

Mickleborough, T.D., **Shei, R-J.**, Stickford, J.L., Wilhite, D.P and Chapman, R.F. Inspiratory muscle training lowers the oxygen cost of breathing. *Journal of Physiology*, 2012. 590(15): 3401 (comments).

ABSTRACTS

Mickleborough, T.D., Vaughn, C.L., Davis, E.M., **Shei, R-J.**, Sinex, J.A., Platt, D., and Wilhite, D.P. Marine lipid fraction PCSO-524™ (Lyprinol®/Omega XL®) attenuates airway inflammation and hyperpnea-induced bronchoconstriction in asthmatic individuals. *Medicine and Science in Sports and Exercise*, 2013. 45(5): S246.

Weavil, J.C., **Shei, R-J.**, Lindley, M.R., and Mickleborough, T.D. Pulmonary adaptations to swim and inspiratory muscle training in sub-elite swimmers. *Medicine and Science in Sports and Exercise*, 2012. 44(5): S395.

MANUSCRIPTS IN PREPARATION

Shei, R-J., Lindley, M.R., and Mickleborough, T.D. Pulmonary adaptations to swim and inspiratory muscle training in sub-elite swimmers.

SERVICE

Indiana University School of Public Health-Bloomington Student Government

Graduate student representative to the School of Public Health-Bloomington Graduate
Studies Committee

Member of 2013-2014 Emissaries for Graduate Student Diversity cohort

School of Public Health-Bloomington Student Ambassadors

GRANTS AWARDED

2012 HPER Student Travel Grant (\$400)

ACADEMIC AWARDS

Indiana University – Bloomington: School of Public Health Fellowship Award

Indiana University – Bloomington: Student Academic Appointment

Indiana University – Bloomington: Dean's Scholarship

PROFESSIONAL SOCIETY MEMBERSHIPS

American College of Sports Medicine

2012-Present

Document date: 27 June 2013